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THE EFFECTS OF LEARNED AUTONOMIC CONTROL ON EXPERIMENTALLY INDUCED FEAR

by



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A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Effects of Learned Autonomic Control on Experimentally Induced Fear" submitted by Harry Wallace Craver Jr. in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

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Abstract

The present study concerns an alternative to Wolpe's technique of systematic desensitization based on relaxation. Wolpe considers that anxiety is due mainly to a "widespread discharge of the autonomic nervous system, and predominantly of its sympathetic division." Recent research has indicated that autonomic responses can be instrumentally conditioned despite a traditional belief to the contrary. If fear is an autonomic response (sympathetic), then training a group of <u>Ss</u> to decrease HR (parasympatheric) should inhibit the fear response. This statement is essentially Wolpe's principal of reciprocal inhibition. The basic assumption of the investigation is that if such autonomic control is possible it would be a more effective way of diminishing fear than through muscular relaxation.

Sixty female first-year nursing students were randomly assigned to the following three groups (G): heart rate training (HRT), relaxation training (RT), and control (C). Over three sessions, there were four phases: adaptation (A), training (T), fear conditioning (FC), and test. During all phases, HR was recorded and between phases, a questionnaire was administered to assess their reactions to the experimental situation. Also, between phases, <u>Ss</u> were asked to report their thoughts and feelings. During the adaptation phase all <u>Ss</u> were allowed to become accustomed to the situation.



During the training phase each group had a different task: HRT <u>S</u>s were to increase or decrease their heart rate to a signal (green light); RT <u>S</u>S were instructed to relax to the signal; and control <u>S</u>s simply counted the number of times the signal occurred. During the fear conditioning phase, half of each G was exposed to a red light paired with a loud noise, while the other half was exposed to a red light only. During the test phases the red light was presented with and without the green light, while the <u>S</u>s were asked to perform the same task as during the training phase.

Both HRT and RT $\underline{S}s$ decreased their heart rate during the green light. While HRT $\underline{S}s$ consistently exhibited greater decreases in heart rate than the RT $\underline{S}s$, the differences between these groups however were only significant during the test phase. There was no evidence of conditioned HR changes in $\underline{S}s$ presented with the loud noise. Nevertheless, these $\underline{S}s$ reported a significant increase in fear. After the test phase, HRT and RT $\underline{S}s$ did not report less fear to the red light than control Ss.

Although HRT and RT <u>Ss</u> could produce heart rate change, this training had little effect on their reported fear. It was suggested that to train people in effective heart rate control it might be necessary to classically condition the HR change. Also, the magnitude of reinforcement is probably important in determining the level of HR change attained.



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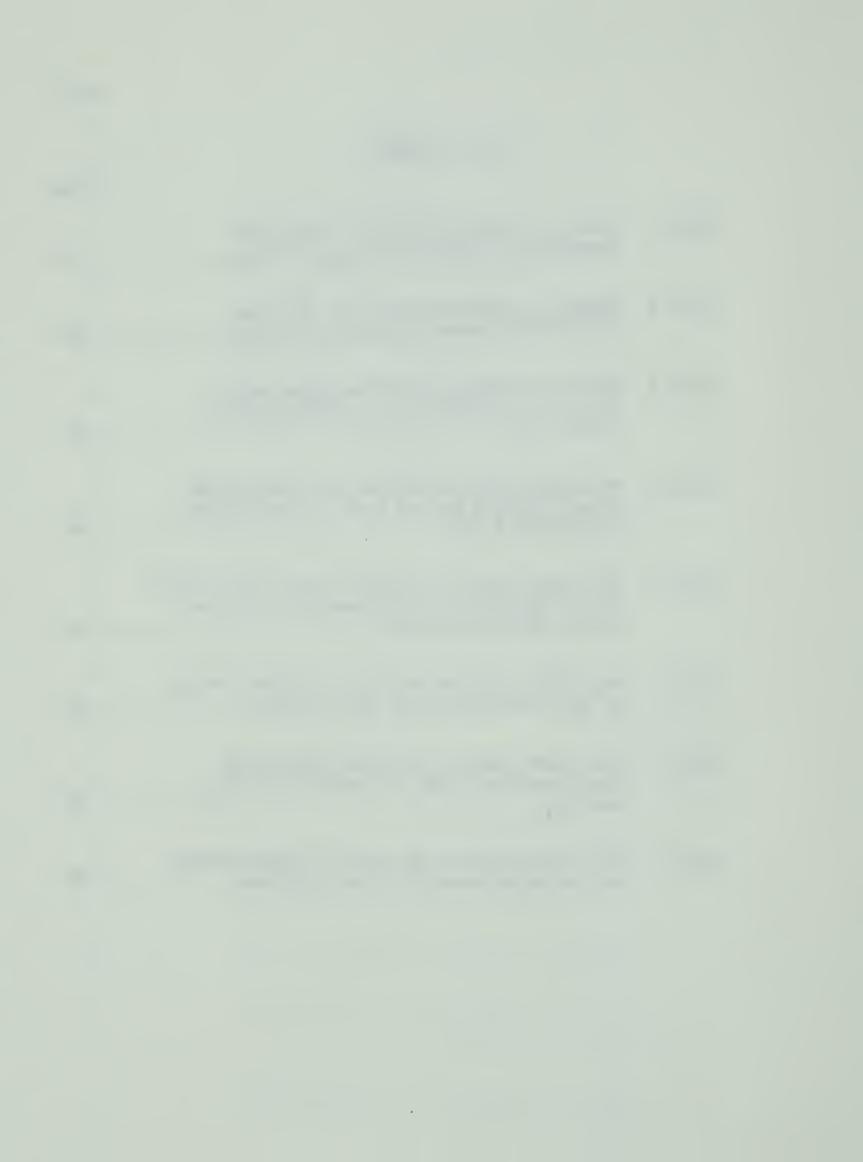


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CHAPTER I

INTRODUCTION

There have been many schools of psychotherapeutic thought concerning the nature of neurosis. Each school has centered on different aspects of the neurotic process. Rogers has emphasized lack of self-fulfillment; Ellis has emphasized irrational thoughts; and Freud emphasized the unconscious. Despite all their efforts, many of the basic tenets are still too nebulous for empirical verification. No researcher has been able to demonstrate that one psychotherapeutic approach can benefit a neurotic patient more than another approach.

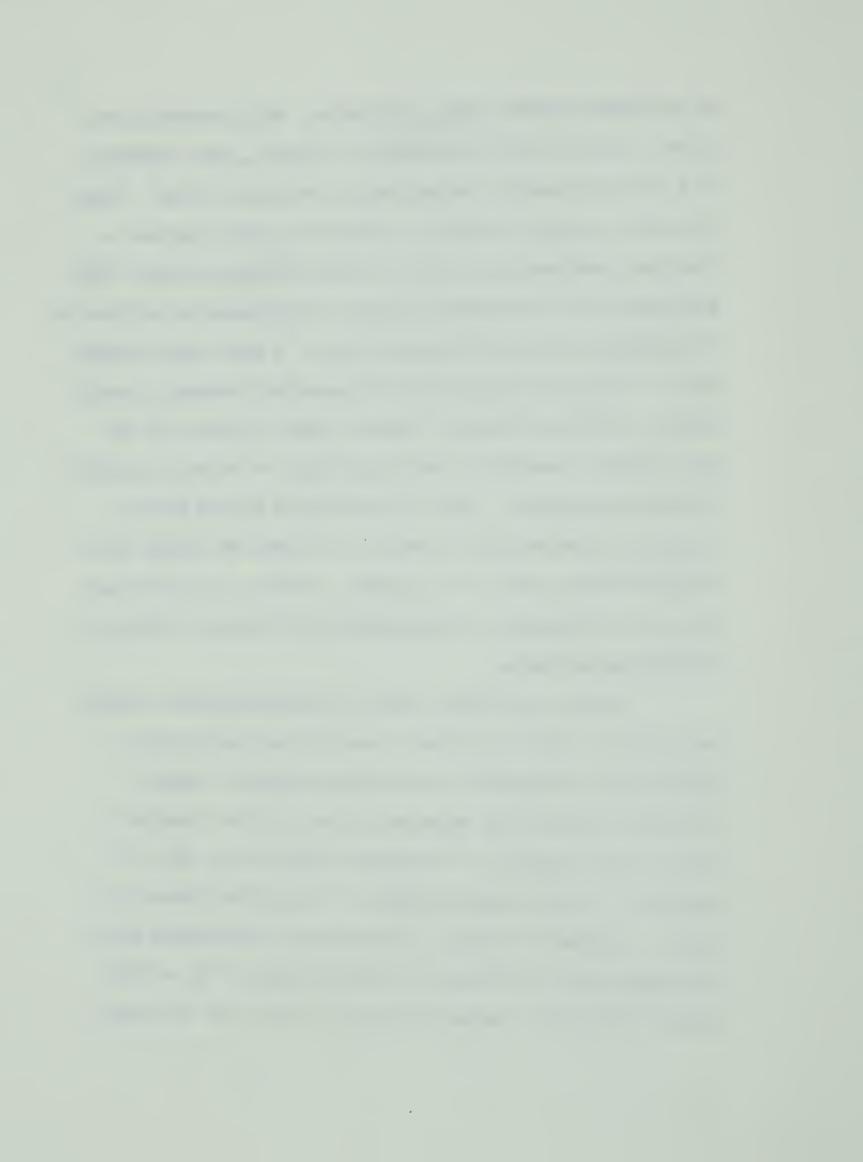
Eysenck (1966), in a summary of studies in the field, found no evidence that any type of psychotherapeutic approach was effective in the treatment of neurosis. Generally speaking, all studies indicate that a comparable percentage of neurotic patients will improve with or without psychotherapy. The onus is with the practitioners of psychotherapy to demonstrate the effectiveness of their approach when applied to neurosis. Many psychotherapists have been distressed by Eysenck's attack on psychotherapy. Some psychotherapists have taken the position that while there was no evidence that psychotherapy helped anyone, there was, also, no evidence that psychotherapy harmed anyone.

Seemingly one of the main obstacles to assessing the benefits of the various schools of therapeutic thought is the lack



of consistency between theory and practice. While therapists may differ in their view of the dynamics of neurosis, their treatment of a particular neurotic patient may be strikingly similar. Strupp found that successful therapists, whether they were Rogerians or Freudians, were markedly similar in their techniques (Strupp, 1955). Regardless of his theoretical position, if the therapist is effective in treatment, he has the characteristics of "a good father" (Strupp, 1955). How these characteristics influence the therapeutic process remains a difficult question. Krasner (1962) believes that the most effective therapist is the therapist who is the most successful "reinforcing machine". That is, the therapist must be able to utilize his "personality" to reward or encourage the changes that he has selected to make in the patient. Krasner's views are exemplary of a new approach to understanding and treating of neurosis called behavior therapy.

The more traditional schools of psychotherapeutic thought emphasize the underlying causes of neurosis and consider many behaviors only "symptoms" of an underlying dynamic. Behavior therapists emphasize the importance of the so-called "symptom". Basically the rationale of the behavior therapists is that all behavior is learned, whether adaptive or maladaptive (mental illness). The behavior therapist centers mainly on observable behavior rather than on unobservable constructs such as "id" or "collective unconscious". Behavior therapists assert that the removal



of a "symptom" effects a "cure" (Ullman and Krasner, 1965). The "symptom" is viewed as a maladaptive or inappropriate behavioral response that has been learned to some cue in the environment. For example, extreme muscular tension might interfere with the correct performance of certain tasks. A young pupil may find himself unable to correctly complete an assignment because of the tension induced by the teacher observing him at his desk. He might be so tense in this curcumstance that the correct response, completing the work, is impossible. From past experiences the pupil might have learned that when his actions have been observed closely, there has been strong disapproval and perhaps punishment. The behavior therapist would seek the stimuli that produced the maladaptive behavior. In this case the teacher's observations would constitute the fear-evoking stimuli that interfere with the pupil's correct performance. To the same stimuli, the behavior therapist would train a response incompatible with tension. For example, the teacher could praise the pupil and give him candy for work well done and ignore incorrect work. The praise and candy might induce a state of relaxation. As the relaxation becomes more and more associated with the teacher, the tension would diminish.

Although Jacobson was not the first individual to consider the therapeutic effects of muscular relaxation, he was the first to develop a systematic application of the technique to many of man's neurotic symptoms. Jacobson conducted several experiments



in which the electrical activity of muscles were recorded (1934). These experiments led him to conclude that certain people do have more difficulty with muscular relaxation than others, and that this difficulty can be associated with mental illness. In his book, Progressive Relaxation, Jacobson (1938) presented a systematic method for acquiring muscular relaxation. During the first session the patient learns how muscular relaxation can benefit him in everyday life. The difference between the sensations associated with muscular relaxation and muscular tension is explained. After the patient grasps this distinction, progressively he learns to induce relaxation in all of the body's muscle groups. The patient uses only the muscles that are necessary for the ongoing activity, thus reducing the overall tension state of the body. For example, while a person is walking, it is not necessary for the arm and facial muscles to be tense. Or, while he is sitting and reading the newspaper, there is no need for face and leg muscles to be tense. The relaxation of all muscles, except those engaged in a specific task, he calls differential relaxation. After months of office sessions and constant practice at home, the patient can begin to substitute muscular relaxation in situations where previously he felt muscular tension. Of patients with a neurotic diagnosis, he reports 21 out of 23 patients were improved either "markedly" or "very markedly".

Early in the 1940's, interest in relaxation was becoming more widespread. Ruesch and Finesinger (1943) found a relationship



between electromyograms and subjects' report of muscular tension. The correlation between subject's report of relaxation and his actual muscle activity were higher for normal subjects than psychiatric and neurological patients. In clinical practice, Fink (1943) developed his own relaxation technique for the treatment of various medical ailments including neurosis. Rathbone (1943) proposed relaxation techniques based on rhythmic and Yoga methods. Yates (1946) considered Jacobson's method entirely too complex, too long, and too costly. Her alternative, "association-set", consisted of repeated associations of the word "calm" with the state of relaxation. Eventually, the patient learns to get "set" to calm himself. Yates claims that her method is quite effective in alleviating problems related to nervous tension.

Jacobson (1947) published a preliminary report concerning the utilization of relaxation methods in the U.S. Navy Air School. He states that 15,000 cadets received instruction which lowered fatigue-tension ratings, reduced percentage of injuries, and produced better sleep as compared with cadets without the relaxation training. As Neufeld (1951) explained, the success of the procedure was quite remarkable in view of the circumstances. The cadets were trained in large groups by officers who received a "crash" course from Jacobson. These officers instructed the cadets one half-hour three times a week for ten weeks. This instruction represented approximately 10% of the usual relaxation procedure.



It was not until ten years later when Wolpe modified Jacobson's technique that relaxation methods began to have a prominent place in psychotherapy. Wolpe has developed clinical techniques based on the principle of reciprocal inhibition for the treatment of neurotic patients. Sherrington (1906) originally intended the term reciprocal inhibition to refer to the inhibition of one spinal reflex by another. Wolpe uses the term to cover all situations where the evocation of one response diminished the strength of a simultaneously occurring response. He suggests that the stronger of two incompatible responses to a stimulus will inhibit the occurrence of the weaker response. When a response is elicited which is incompatible with the anxiety response to a particular situation, the bond between that situation and the anxiety response is weakened.

One of Wolpe's methods of treating phobic behavior is termed systematic desensitization based on relaxation. The procedure is as follows: The person is taught muscular relaxation which takes 7-10 sessions with practice at home. Jacobson, it may be noted, required 100-200 sessions to teach relaxation. Jacobson never emphasized a process in which tension was used to learn relaxation. In contrast, Wolpe had patients induce tension as well as relaxation in order to learn the differences in sensations between the two states. An example of this is clenching the right fist for 30 seconds and then relaxing it in order to compare the sensations associated with relaxation and tension. While Jacobson was not directly concerned



with the tension-evoking stimuli, which may occur in the life situation, Wolpe attempts to decrease the tension associated with the life situation in a more controlled office setting. A list of anxiety-evoking situations related to the phobia is arranged in a hierarchy from the least to the most anxiety-evoking item. The hierarchy is taken from the patients' "homework" which consists of a "list of everything he can think of that is capable of frightening, distressing, or embarrassing him in any way, excepting of course, situations that would frighten anybody, such as meeting a hungry lion".

After the patient is instructed to relax as deeply as possible, sometimes aided by hypnosis, the phobia is aroused. He is asked to imagine a neutral scene followed by the least anxiety-evoking item in the hierarchy. If the phobia was of crowds, the hierarchy might start with being with a good friend and walking on an unpopulated street. The last item might be going to a party and meeting new people. The patient is instructed to raise his hand at the occurrence of any tension associated with the scenes in the hierarchy. If anxiety occurs, the scene is terminated immediately.

The importance of the relaxation and accurate construction of the hierarchy cannot be minimized. If the relaxation is not complete or, if the hierarchy is not graduated properly, anxiety may be heightened instead of lessened. During the session the



patient is associating the response of relaxation to the stimuli which were previously anxiety-eliciting. Wolpe hypothesizes that since the response of relaxation reciprocally inhibits the anxiety response, the phobic reaction is eliminated. The number of sessions with item presentations vary greatly, but are usually between 10 and 25. The therapy is successful when the patient is no longer manifesting anxiety to the items on the hierarchy, and subsequently, to the relevant stimuli in the real world.

Wolpe has reported a higher success rate than is typical of the more traditional psychotherapy. His main involvement has been with phobic patients. Since most psychotherapeutic approaches have found phobia one of the most difficult of neuroses to treat effectively, his success rate is remarkable.

Wolpe makes certain assumptions about the nature of anxiety experienced by neurotic patients. He defines anxiety as:

is meant the autonomic response pattern or patterns that are characteristically part of the organism's response to noxious stimulation. A noxious stimulus is one that causes tissue disturbance of a kind that tends to lead to avoidance responses. When noxious stimulation itself evokes the anxiety responses, it is through nerve channels formed in the normal course of biological development of the organism. This is unconditioned (unlearned) anxiety.... A stimulus not previously capable of evoking anxiety may acquire the power to do so if it happens to be acting on the organism when anxiety is evoked by another stimulus. (p. 34)

This passage presents a learning theory interpretation of anxiety based on a classical conditioning paradigm. The actual source of



the anxiety is attributable to a strong autonomic discharge, predominantly of the sympathetic division. This discharge is manifest in increased heart rate (HR), respiration, blood pressure, and other physiological changes (Wolpe, 1958).

Wolpe's usage of the term "anxiety" is by no means a commonly accepted definition. Unfortunately, the word "anxiety" is defined quite differently by different authors. According to English and English (1958):

When a term is frequently employed in behavioristic learning theory, in psychoanalysis, and in nearly every field of psychology between them, the variety and shadings of meaning become very troublesome. Anxiety must be read with great vigilance for the author's meaning or, more often than not, his several meanings.

After reviewing the literature concerning "anxiety", Maher (1966) came to these conclusions: "Anxiety is best regarded as a construct or inferred state... Measures of anxiety in the three categories (verbal report, overt (motor) behavior, and physiological measurement) have not generally produced high correlations with each other, and many problems exist with regard to their reliability." Cattell and Scheier (1961) state that the basic operational definitions of anxiety stem from physiological measures, overt behavior, and self-report. While Wolpe utilizes overt behavior and self-report in diagnosis and treatment, his theoretical position is essentially physiological as pointed out above.

The technique of muscular relaxation has focused on the



relaxation of the skeletal muscles. If anxiety is diminished by Wolpe's relaxation technique, it is logical to assume then, relaxation must affect autonomic responses. There is experimental evidence that decreased muscle tension is accompanied by decreased heart rate (Jacobson, 1940). One could therefore assume that if anxiety is basically an autonomic reaction, then the most effective way to reduce anxiety might be to directly treat the autonomic dysfunction. The goal of psychotherapy might then be conceived as reconditioning the autonomic responses associated with anxiety.

Recent research has indicated that autonomic responses can be instrumentally conditioned despite a traditional belief to the contrary (Shearn, 1962; Engel and Hansen, 1966; Engel and Chism, 1967; Hnatiow and Lang, 1965; Miller, 1969). The old idea that autonomic responses are involuntary and thus not applicable to instrumental conditioning is being revised. Brener and Hothersall (1966) suggest that "where a given response falls on the voluntary/involuntary continuum, is the availability of specific feedback from the response in question." Skeletal muscle have been conditioned instrumentally. The response of these muscles provide considerably more feedback than autonomic response.

With autonomic control now demonstrated, the question remains whether or not it can be utilized in a clinical setting. Ascough and Sipprelle (1968) view Wolpe's psychotherapy by reciprocal in-

hibition "as an attempt to manipulate or to decrease certain



conditioned autonomic responses by verbal conditioning." On the basis of these researchers' findings, which involve heart rate control by verbal reinforcement, they conclude that "certain behavior modification techniques...may accomplish behavior modification by directly influencing autonomic responses constituting anxiety."

Since a central goal of psychotherapy is to reduce anxiety augmented feedback from organs innervated by the autonomic nervous system might be used in a process designed to faciliate anxiety reduction.

Instead of implementing muscular relaxation with systematic desensitization, reduction of sympathetic activity could possibly be brought about through appropriate training procedures, and then associated with the anxiety-evoking stimuli. The purpose of the present experiment is to determine if such a procedure, as outlined above, is indeed possible. Briefly stated, the investigation is designed to determine whether an instrumentally acquired decrease in heart rate can reciprocally inhibit anxiety.

More specifically, this study will be concerned with the following question: Can training in HR reduction produce a greater decrease in HR than acquired muscular relaxation or simply being exposed to the experimental situation? And, related to the preceding, is training in HR reduction a more effective means for



eliminating anxiety than instructed relaxation or exposure to the experimental situation? This project should not be construed as an attempted comparison between autonomic training and Wolpe's method of teaching relaxation. In the actual study the relaxation subjects (<u>S</u>s) were requested to relax, but they had no instruction in relaxation techniques.



METHOD

Subjects

The subjects ($\underline{S}s$) consisted of 72 female first-year nursing students at the University of Alberta Hospital. The experimenter (\underline{E}) met with students during their formal class period. A general description of the study was presented including the possibility of $\underline{S}s$ hearing a loud noise. Those who expressed an interest either signed up for prearranged time or left their name and telephone number for later contact. Certain restrictions were placed upon their participation: All $\underline{S}s$ who had a recent history of cardiac, respiratory, auditory, or psychological problems were eliminated. The volunteers ranged in age from 17 to 26 years. Of the original sample, $\underline{S}s$ were statistically treated. Six $\underline{S}s$ were pilot $\underline{S}s$; four produced unreliable HR recordings; and two $\underline{S}s$ failed to complete the three sessions.

Apparatus

The heart measure was obtained by Onyx EKG leads placed on the superior part of the sternum and the left inferior rib cage. The signal from the electrodes was transmitted via an Onyx FM telemetry System (Model 3.002) to a cardiotachograph preamplifer (Model 5P4D) of a Grass Model 5D polygraph. From the output of the



cardiotachograph, a modified Beckman cardiotachometer coupler was connected. The coupler provided a DC potential which was directly proportional to the frequency of the heart beat. This potential was registered on a meter located on a panel in front of the subject. This meter indicated one unit change for each three beats. Grason-Stadler counters (E3700A, with stepper-E3129A and electronic timer-EllOOH) were connected to the output of the coupler and provided a cumulative record of heart beats. Respiration was measured by the use of a chest bellows attached to a strain guage (Statham transducer Model P25BB), the output of which was amplified by a low level DC pre-amplifier (Grass 5PIK). In addition to the meter, the panel contained a red and green light. A random noise generator (General Radio Co., 1390-B) amplified by a 12-Watt P.A. amplifier was used to produce white noise. The time durations for each of the two lights and the white noise were controlled by three Hunter Timers. A tape recorder was employed to present the Ss' instructions.

Procedure

The data were collected in the Psychiatric Laboratory of the Clinical Sciences Building, University of Alberta, Edmonton, Alberta. The 9 X 12 room was electrically and acoustically shielded. With the display panel before her, \underline{S} was seated in a comfortable chair. The testing of each \underline{S} required three sessions and was usually completed within a week. Over the three sessions there were four phases.



Session I

Adaptation Period (10 min.) (phase A)

First Training Period (24 min. (phase T₁)

Session II

Second Training Period (24 min.) (phase T_2)

Session III

Third Training Period (12 min.) (T_3)

Fear Conditioning (11 min.) (FC)

Test phase (18 min.)

Before phase A, \underline{S} was asked to sign a permission slip (Appendix A) and answer a few questions about their physical state. The following taped instructions were then delivered to \underline{S} .

There is nothing difficult about this experiment. You may be asked just to experience the occurrence of a light or perform a simple task. It is not designed to assess personality or intelligence. The study in which you are participating involves four phases and takes three sessions to complete. Before each phase, you will be told what to expect or what the phase involves. The purpose of the experiment is to assess certain bodily reactions, these reactions you may or may not be aware of, under several different conditions. During the course of the study you will be called upon to make certain assessments of your thoughts and feelings. These will become clear as the occasion arises.

If there were no questions, the recording devices were attached to the \underline{S} . Instructions for phase A were given as follows:

This phase is to allow you to become accustomed to the situation. It is important that during this stage and all other stages for you to minimize your movements. At the beginning and conclusions of this phase, you will be asked to report some of your feelings and thoughts. The



key objective in phase A is to relax, and breathe normally. The phase will last approximately 10 minutes. The lights will be dimmed to facilitate your relaxation. Also, you will see some lights on the panel before you. It is not important to count these lights or to recognize any pattern. The lights are present to help your body adapt to the situation.

Again, \underline{S} had an opportunity to ask any questions. At this time \underline{S} was asked: "Is there any particular reason this experimental setting should upset you?" Then \underline{S} was given an Experimental Situation Questionnaire (ESQ) (Appendix B) in order to assess some of her reactions during each phase. The ESQ was administered before and after each phase or part of phase T.

After a short delay for equipment adjustment, phase A was begun. The lights were dimmed and the experimenter (\underline{E}) was situated directly behind \underline{S} in the partitioned equipment area. Heart rate was recorded during the first, fifth, and ninth minutes to obtain a resting heart rate. While these three one-minute periods were in effect, no room lights were on; but between these periods the green and red lights would appear sometimes simultaneously and sometimes separately.

All $\underline{S}s$ were exposed to phase A. Phase T involved placing $\underline{S}s$ in one of three groups: Heart Rate Training (HRT), Relaxation Training (RT), and Control (C). Each group consisted of 20 subjects. Assignment to each group was determined randomly. During T_1 and T_2 all $\underline{S}s$ were exposed to 20 occurrences of the green light. During



T₃ all <u>Ss</u> were exposed to 10 occurrences of the green light.

Each occurrence of the green light was separated by either a 30,

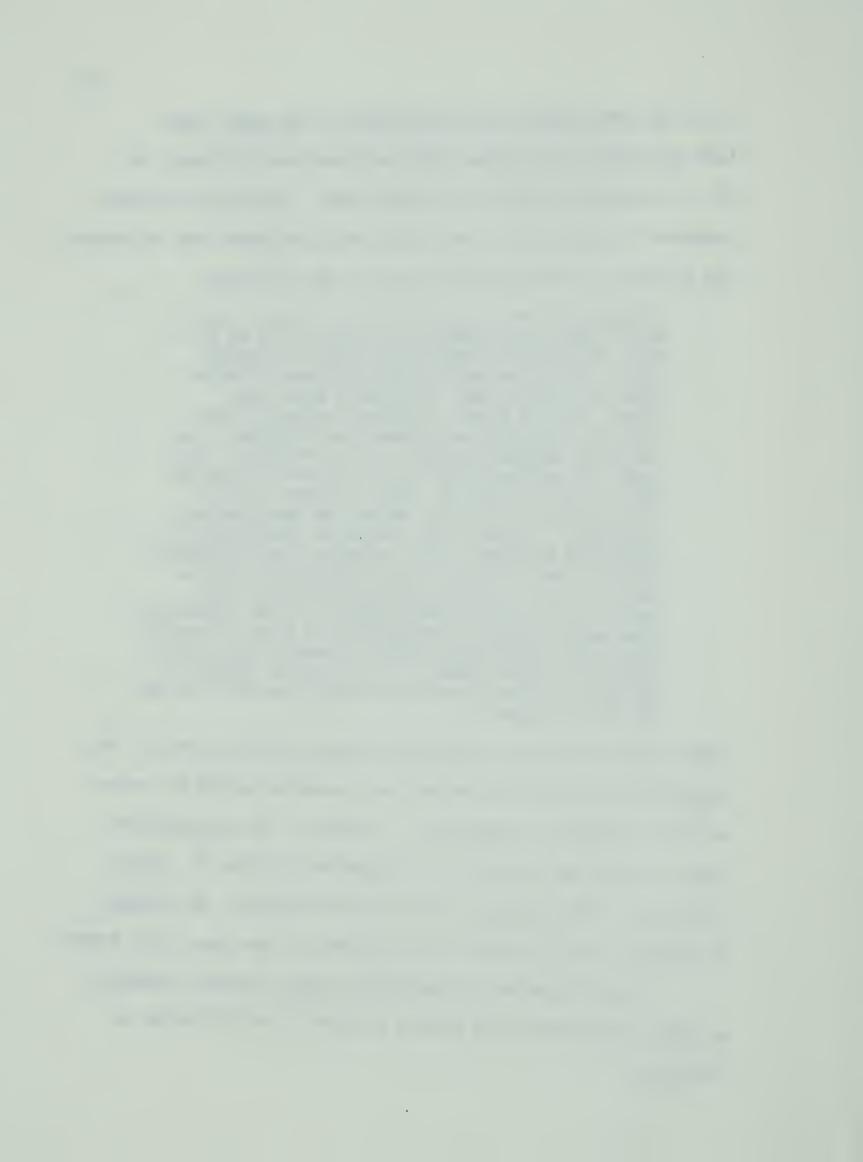
45, or 60-second interval in a random order. During the 30-second exposure to a green light, each group had a different task to perform.

The following instructions were given to the HRT group.

The meter before you will monitor your heart rate. When a green light comes on your task will be to keep the pointer of the meter below a designated level. Watch the meter closely because a fluctuation of just one unit indicates a heart rate change of three beats. Although the bodily feelings of relaxation and calmness are important, use the meter to gauge how well you are relaxing: the lower the reading of the meter, the more successful you will be at relaxing. When the green light is off, your task will be to return the meter reading designated. Possibly the thought of increasing or decreasing your heart rate is against the traditional notion of heart rate being an involuntary response. Recent research, however, had demonstrated that people can vary their heart rate; it is not impossible. Some people show great fluctuation in heart rate while others show little fluctuation in heart rate. Either reaction is quite normal. It also may be helpful if you do not actively pursue this task, but just sit back and let it happen.

These instructions were repeated before each training period. The second and third training periods were conducted during the second and third sessions, respectively. In phase T, the designated HR level for each was his initial HR determined in phase A. Essentially, Ss in HRT group were learning two responses: HR decrease to the green light present, and HR increase to the green light absent.

The RT <u>Ss</u> were also requested to make different responses to green light present and absent, as shown in the following instructions.



When the green light comes on your task will be to allow yourself to relax. That is, to pay attention to your mental and physical state and try to induce a feeling of calmness and tranquility. It is quite important to relax when the green light is on, but only when the green light is on. When the green light is off, your task will be to clench your right fist tightly. It may be helpful if while the green light is on you do not actively pursue relaxation, but just sit back and let it happen.

The control group was exposed to the same sequence of green lights as the other two groups. In this case, however, the control group was given the following instructions:

When the green light comes on your task will be to remain alert and attend to the light. You are in a comparison group that will assess the effect of being in this situation over a period of time. Count how many times the green light occurs. This counting is simply to check how alert you remain.

The meter was visible to the RT and C Ss, but it was not in operation.

After <u>S</u> had completed the third part of phase T, she was introduced to the third phase, the fear condition. During this phase (FC), half of <u>S</u>s (group RL + N) received white noise paired with the red light, the conditioned stimulus (CS), on a 50% partial reinforcement schedule. The other half of the <u>S</u>s (group RL) received the CS only. The white noise (US) was delivered through the earphones at an intensity of 105db, and a duration of one second. During all phases, <u>S</u>s were wearing earphones. The CS was presented for five seconds, during which time the US occurred at either the first, third, or fifth second of that period. Variation of US was



introduced to guard against possible habituation to the US. The CS occurred 20 times with the US paired on 10 occasions. Time interval between CSs was either 15, 30, or 45 seconds. Instructions for phase FC were as follows:

During this phase you will see a red light on the panel before you (for the fear condition: you will also hear a noise through the earphones). The light will come on periodically; simply note its occurrence. You do not have to count them or recognize any pattern.

The test phase was designed to assess the effects of phases T and FC. The test included 15 30-second intervals of green light in precisely the same order as the first 15 intervals of phase T. On ten occasions the red light was presented 15 seconds after the onset of the green light. In addition, the red light was presented on 10 occasions 15 seconds after the cessation of the green light. The $\underline{S}s'$ instructions were as follows:

During this phase you will experience both the red and green lights. When the green light is on, it will have the same significance as it did for phase T. This phase is to assess your reaction to the lights.

In the case of the HRT $\underline{S}s$, no HR feedback was presented. After completing the task, $\underline{S}s$ filled out the questionnaire (ESQ) for the last time. Then $\underline{S}s$ were shown their physiological responses and were given an explanation of the experiment. Before leaving, each \underline{S} was requested not to reveal to anyone the nature of the experiment.



Experimental Design

The independent variables were the group (G) factor (HRT, RT, and C groups); the fear conditioning (FC) factor (Ss receiving red light and noise [RL + N], and Ss receiving only the red light [RL_{only}]; the stimulus factor (green light [GL] and/or red light [RL]); trials (T); and the periods (P) factor (heart rate was measured over successive 7.5 second periods).

Except for the test phase, HR was analyzed for each phase (and the three parts of phase T), by a 4-factor analysis of variance (ANOV) for repeated measures. The test phase was analyzed by a 5-factor ANOV for repeated measures. Each item of the ESQ was interpreted by a 3-factor ANOV for repeated measures: G, FC, and RA (repeated administrations). The number of administrations depended on the particular item. Since respiration was reliably recorded for only one-third of \underline{S} s, this measure was discarded. Because of the large number of ANOV and F ratios computed, it was considered prudent to use only the .01 level of significance.



CHAPTER III

RESULTS

Pre-Phase Questionnaire

Since physiological measures were recorded, an attempt was made to assure that no prior conditions or circumstances would influence the results. The information sheet was this safeguard. Upon examination there were no questions on which <u>Ss</u> greatly differed. <u>Ss</u> rated their health as either good or excellent, and they reported no recent history of cardiac, respiratory, hearing, or psychological problems. Except for two <u>Ss</u> who were taking birth control pills, <u>Ss</u> reported that they were free from drug use. Only three <u>Ss</u> reported beginning their menstrual cycle. Most <u>Ss</u> considered themselves physically fit, and only five <u>Ss</u> smoked (smoking was prohibited within 30 minutes prior to each session). Stimulants, such as coffee or tea, were not taken prior to each session.

Once the information was taken, each \underline{S} was asked, "Is there any reason this experimental setting should upset you?" Only one \underline{S} expressed concern which, with reassurance, dissipated at the end of phase A. The three types of data (HR, ESQ, and SR) are considered separately beginning with HR. All analyses of variance tables for HR and ESQ are found in Appendices C and D respectively.



Heart Rate

Phase A: This analysis was designed to determine whether there were any differences between the six experimental groups (G, FC) prior to the introduction of any differential treatment. The G and FC \underline{S} s were compared for the eight consecutive periods of the first, fifth, and ninth minutes $(3 \times 2 \times \underline{8} \times \underline{3})^{1}$. None of the F ratios approached significance. Seemingly all \underline{S} s were relaxed before phase A began. Except for one \underline{S} who had an initial HR over 100 beats/minute, \underline{S} s had relatively stable HRs.

Phase \underline{T}_1 : This phase was analyzed for the three subject conditions (G), 20 Trials (T), the occurrence of the green light (GL), and the four 7.5 second periods (P) under GL_{on} and GL_{off} (3 X 20 X $\underline{2}$ X $\underline{4}$). The statistical treatment of this part of phase T and the following two parts (2, 3) is an attempt to determine whether \underline{S} s could control their HRs. And would their heart rate changes be greater than that attained by instructed relaxation or simply experiencing the experimental situation?

Heart rate for GL_{on} was lower than GL_{off} (\underline{F} = 14.32, \underline{df} = 1/57, \underline{p} < .01). The interaction between groups and green light was significant (\underline{F} = 5.83, \underline{df} = 2/57, \underline{p} < .01). As shown in Table 1, HRT \underline{S} s exhibited greater heart rate differences between GL_{on} and

The numerical notation designates the number of levels under each factor. This designation follows the order of the factors presented in the sentence. The underlining indicates the repeated factors.



GL_{off} than RT <u>Ss</u> in the expected direction. Control <u>Ss</u> showed a tendency in the opposite direction. The difference for the control <u>Ss</u> differed significantly from the difference for the other two groups, (Tukey (b), $\underline{q} = .22$, $\underline{p} < .01$, Winer, 1962).

Phase \underline{T}_2 : This phase was analyzed in precisely the same manner as phase \underline{T}_1 . Again, heart rate for \underline{GL}_{off} was higher than \underline{GL}_{on} (\underline{F} = 12.12, \underline{df} = 1/57, \underline{p} < .01). The interaction between groups and green light was significant (\underline{F} = 12.97, \underline{df} = 2/57, \underline{p} < .01, Table 2). HRT and RT \underline{S}_s exhibited greater HRs during \underline{GL}_{off} than \underline{GL}_{on} , wheras control \underline{S}_s showed a tendency in the opposite direction. The difference for the control \underline{S}_s differed significantly from the difference for the other two groups (Tukey (b), \underline{q} = .12, \underline{p} < .01).

Phase T₃: The same analysis as phase T₁ and T₂ was applied except there were only 10 Trials. The interaction between G, T, and GL was significant (\underline{F} = 3.52, \underline{df} = 18/513, \underline{p} < .01). As Table 3 shows the HRT \underline{S} s consistently showed reduced HR during the green light, whereas, the performance of the RT \underline{S} s was more erratic. The G X GL X T X P interaction was also significant (\underline{F} = 1.57, \underline{df} = 54/1539, \underline{p} < .01, Appendix E). Since there were no systematic effects apparent in the means for this interaction, an interpretation will not be attempted.

Phase FC: This phase was analyzed for G, FC, the 20 trials, and the two periods (P) of 7.5 seconds in each trial (3 X 2 X $\underline{20}$ X $\underline{2}$). The interaction between FC and P was significant (\underline{F} = 10.10, \underline{df} = 1/54, \underline{p} < .01). As shown in Table 4, RL_{only} \underline{S} s had a higher HR during



Table 1: Phase T₁

Mean Heart Rate Per 7.5 Seconds For the Three Experimental Groups During ${\rm GL}_{\rm on}$ and ${\rm GL}_{\rm off}$

	GLon	GLoff	Differences		
HRT	10.29	10.60	+.31		
RT	10.25	10.44	+.19		
С	10.61	10.59	02		

Table 2: Phase T₂

	GLon	GL _{off}	Differences
HRT	10.07	10.28	+.21
RT	10.05	10.25	+.20
С	10.81	10.71	10



Table 3: Phase T_3 Mean Heart Rate For The Three Experimental Groups

As A Function Of GL_{on} and GL_{off} And Trials (T)

HRT					RT		С		
Trial	s GL _{on}	GLoff	Differ- ences	GLon	GL _{off}	Differ- ences	GLon	GL _{off}	Differ- ences
٦.	10.10	10.16	+.15	10.36	10.35	01	10.56	10.60	+.04
2.	10.26	10.30	+.04	10.89	10.35	54	10.75	10.73	02
3.	10.20	10.23	+.03	10.51	10.18	33	10.89	10.83	06
4.	10.09	10.31	+.22	10.38	10.36	02	11.08	10.69	39
5.	10.21	10.53	+.32	10.13	10.35	+.22	10.75	10.73	02
6.	10.01	10.49	+.48	9.20	8.16	04	10.81	10.78	03
7.	10.06	10.48	+.42	8.23	8.26	+.03	10.90	10.56	34
8.	10.14	10.34	+.20	8.33	8.25	08	10.93	10.95	+.02
9.	10.03	10.34	+.31	8.20	8.18	02	10.74	10.77	+.03
10.	10.21	10.28	+.07	8.09	10.08	+1.99	10.79	10.60	19



Table 4: Phase FC

Mean Heart Rate For The Two Fear Conditioning Groups Prior To The Red Light (P_1) And During The Red Light (P_2)

	P ₁	P ₂	Differences
RLonly	10.30	10.40	+.10
RL + N	10.20	9.94	26

Table 5

Mean Heart Rate For The Noise And No Noise Trials Of RL + RN $\underline{S}s$ Prior To The Red Light (P₁) And During The Red Light (P₂)

	P ₁	P ₂	Differences
Noise	10.27	9.84	43
No Noise	10.13	10.04	09



the red light (P_2) than preceding it (P_1) . For the RL + N $\underline{S}s$, this relationship was reversed. An ANOV comparing the RL + N $\underline{S}s$ on the 10 trials of no noise showed a significant noise by period interaction $(\underline{F}=8.22,\underline{df}=1/27,\underline{p}<.01,$ Table 5). Apparently this interaction was primarily due to a reduction in HR on trials when the red light was followed by noise. It should be noted that there was little difference between the means for trials without noise.

Test Phase: This phase was analyzed in two parts. Both analyses consisted of a 5-factor ANOV for repeated measures which included the group factor (G); the fear conditioning factor (FC); stimuls factor (GL); Trials (T); and the period (P) factor (3 X 2 X $\underline{2}$ X $\underline{10}$ X 4). The red light (RL) occurred randomly on 10 of the 15 occasions of GL $_{on}$ and GL $_{off}$. The data for the first analysis consisted of only those presentations of GL $_{on}$ and GL $_{off}$ during which RL occurred. The second analysis included the 5 out of 15 presentations of GL $_{on}$ and GL $_{off}$ when the red light was not present (3 X 2 X $\underline{2}$ X $\underline{5}$ X $\underline{4}$).

The First Analysis (a)

During the 7.5 second period in which the red light occurred, heart rate was significantly lower than the immediately preceding and following periods ($\underline{F} = 5.01$, $\underline{df} = 3/62$, $\underline{p} < .01$, Table 6). The interaction between the G and GL was significant ($\underline{F} = 9.44$, $\underline{df} = 2/54$, $\underline{p} < .01$). HRT $\underline{S}s$ showed the greater heart rate difference



Table 6

Mean Heart Rate For The Four 7.5 Second Periods
Of The First Analysis Of The Test Phase

Periods	1	2	3	4
	10.22	10.21	10.11	10.24

Table 7: Test Phase (a)

Mean Heart Rate For The Three Experimental Groups When Red Light Is Present During $\operatorname{GL}_{\operatorname{on}}$ And $\operatorname{GL}_{\operatorname{off}}$

	GLon	GL _{off}	Differences
HRT	9.92	10.18	+.26
RT	9.98	10.11	+.13
С	10.55	10.42	13



between GL_{on} and GL_{off} than the RT $\underline{S}s$ (Table 7). Control $\underline{S}s$, again, had a greater HR for GL_{on} than for GL_{off} . The difference between GL_{on} and GL_{off} for each group differed significantly from the difference for every other group (Tukey (b), \underline{q} = .04, \underline{p} < .01). The G X GL X P interaction was significant (\underline{F} = 3.17, \underline{df} = 6/162, \underline{p} < .01, Figure 1). The difference between GL_{on} and GL_{off} increased over periods for HRT $\underline{S}s$ while it tended to decrease for the other two groups. Also, both RT and HRT $\underline{S}s$ showed the greatest difference during the occurrence of the red light (P_3). None of the interactions with FC approached the .01 level of significance.

The Second Analysis (b)

This analysis consisted of the five blocks of time when GL_{on} and off occurred in the absence of the red light. There was a significant heart rate change over the 7.5 periods ($\underline{F} = 7.75$, $\underline{df} = 3/162$, $\underline{p} < .01$, Table 8). The minimum HR was during P_4 unlike the previous analysis in which the minimum HR was during P_3 .

The G X FC X T X P interaction was significant (\underline{F} = 1.84, \underline{df} = 24/648, \underline{p} < .01). Since no systematic effects were apparent, no interpretation will be attempted (Appendix F).

Experimental Situation Questionnaire (ESQ)

The response for each question ranged on a scale from 1-9, with 1 the lowest and 9 the highest.

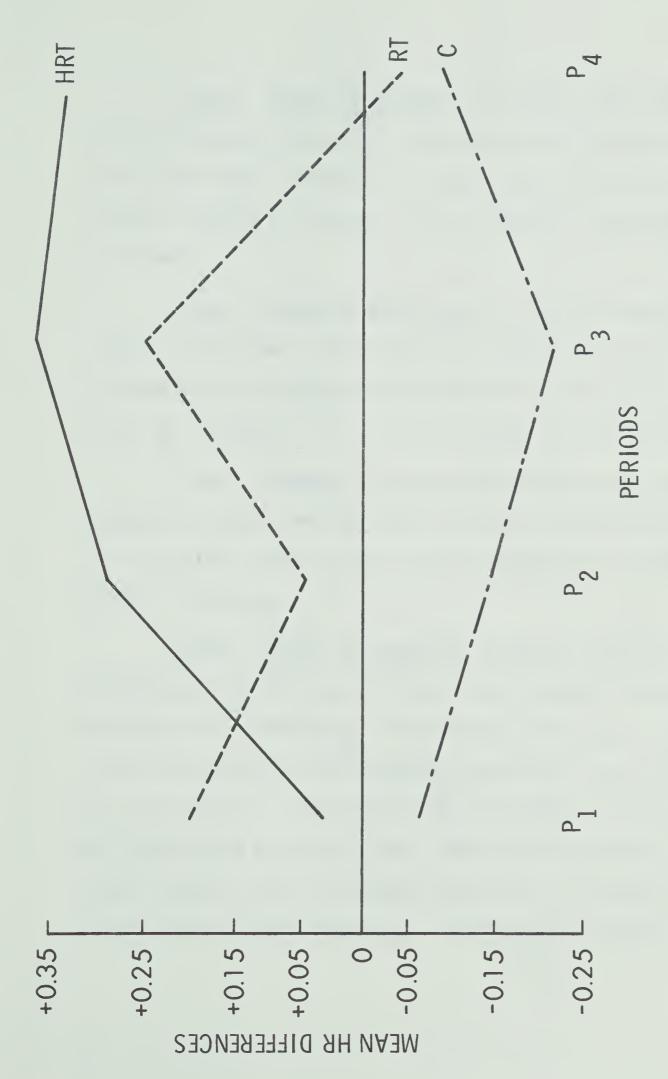


Table 8

Mean Heart Rate For The Four 7.5 Second Periods
Of The Second Analysis Of The Test Phase

Periods 1 2 3 4 10.26 10.30 10.20 10.11





7.5 second periods (P). P₁ and P₂ were the periods immediately prior to the and GL_{OFF} for the three experimental groups as a function of successive The First Analysis of the Test Phase. Mean HR differences between GLON FIGURE 1:

occurrence of the RL (P_3) , while P_4 immediately followed RL.



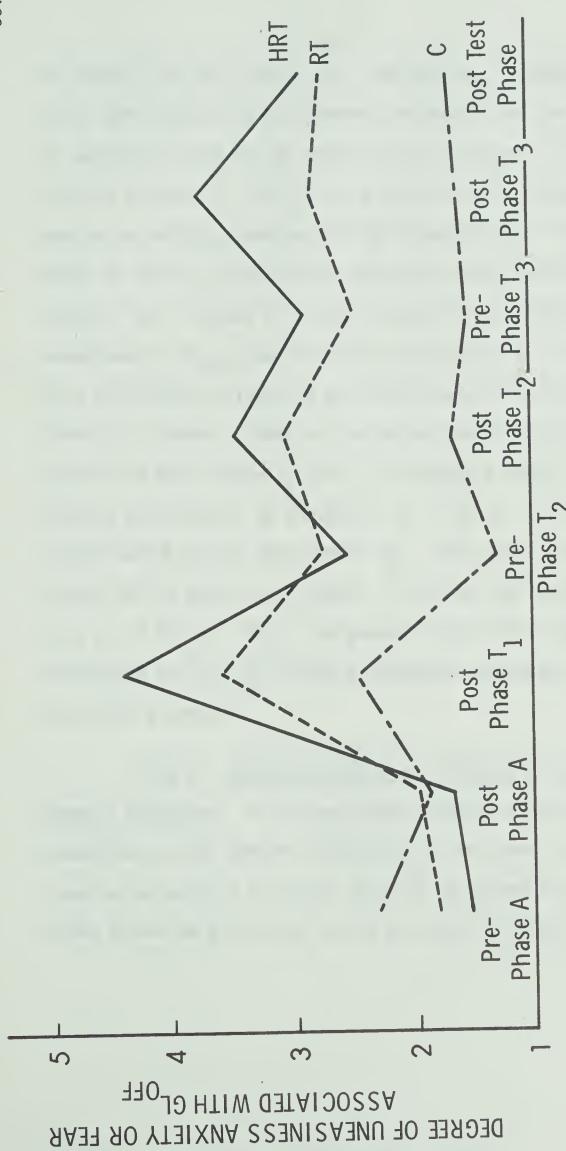
Item 1: <u>Degree of Interest</u>: This item, like Items 2, 7, and 8, attempted to assess <u>S</u>s' overall motivation and attentiveness to the experiment. There were no significant effects resulting from the analysis of variance. All <u>S</u>s expressed a moderate level of interest.

Item 2: Degree of Restlessness: This item concerned the level of restlessness (distractibility) experienced by the $\underline{S}s$. Although there was <u>variation</u> over administration for all $\underline{S}s$, (\underline{F} = 11.59, $\underline{d}f$ = 8/432, \underline{p} < .01), the three groups did not differ.

Item 3: Degree of Uneasiness, Anxiety or Fear associated with GL_{on} : While there was some variation over administration ($\underline{F} = 5.78$, $\underline{df} = 7/378$, $\underline{p} < .01$), all \underline{S} s reported a low level of anxiety to the GL_{on} .

Item 4: Degree of Uneasiness, Anxiety, or Fear associated with $\underline{\mathsf{GL}}_{\mathsf{Off}}$: This item concerned the level of general uneasiness that $\underline{\mathsf{Ss}}$ may have experienced in the absence of the green light. The interaction between G and RA was significant ($\underline{\mathsf{F}}$ = 3.08, $\underline{\mathsf{df}}$ = 14/378, $\underline{\mathsf{p}}$ < .01, Figure 2). HRT $\underline{\mathsf{Ss}}$ and RT $\underline{\mathsf{Ss}}$ were asked to perform tasks of increasing HR and tensing their right hand respectively. These groups reported greater uneasiness than control $\underline{\mathsf{Ss}}$ who had no particular task associated with $\mathsf{GL}_{\mathsf{off}}$. Control $\underline{\mathsf{Ss}}$ had the same level





REPEATED ADMINISTRATIONS

Degree of Uneasiness, Anxiety, or Fear Associated with GLOFF FIGURE 2: Mean responses of three experimental groups to ESQ Item 4: as a function of repeated administration.



of anxiety for this item as for Item #3, GLon. Statistical tests were applied to each administration of the item in order to determine where the Gs significantly differed. At the conclusion of phase T_1 , HRT $\underline{S}s$ had a significantly higher level of anxiety with GL_{off} than control <u>S</u>s (Tukey(a), $\underline{q} = 1.20$, $\underline{p} < .05$). Prior to phase T₂, each group had their lowest anxiety since phase A; but, HRT and RT Ss had a significantly higher level of uneasiness to GL_{off} than control <u>S</u>s (Tukey(a), $\underline{q} = 1.20$, $\underline{p} < .05$). This significant difference was maintained at the conclusion of phase T_2 ; however, there was increased anxiety for all \underline{Ss} . Both before and after phase T_3 , HRT $\underline{S}s$ expressed a higher level of anxiety than control $\underline{S}s$ (Tukey(a), \underline{q} = 1.20, \underline{p} < .05). RT $\underline{S}s$ were significantly higher than control Ss. Upon conclusion of the test phase, HRT Ss again, were higher in anxiety than control Ss (Tukey (b), $\underline{q} = 1.20$, $\underline{p} < .05$). The general trend for all \underline{S} s, but most pronounced for HRT Ss, was more expressed uneasiness just after than before phase T.

Item 5: Degree of Uneasiness, Anxiety or Fear in the General Situation: All three groups reported a low degree of uneasiness to the general situation. So who were assigned to receive noise (RL + N) during phase FC responded differently from those So who were assigned to the no noise condition (RL only)

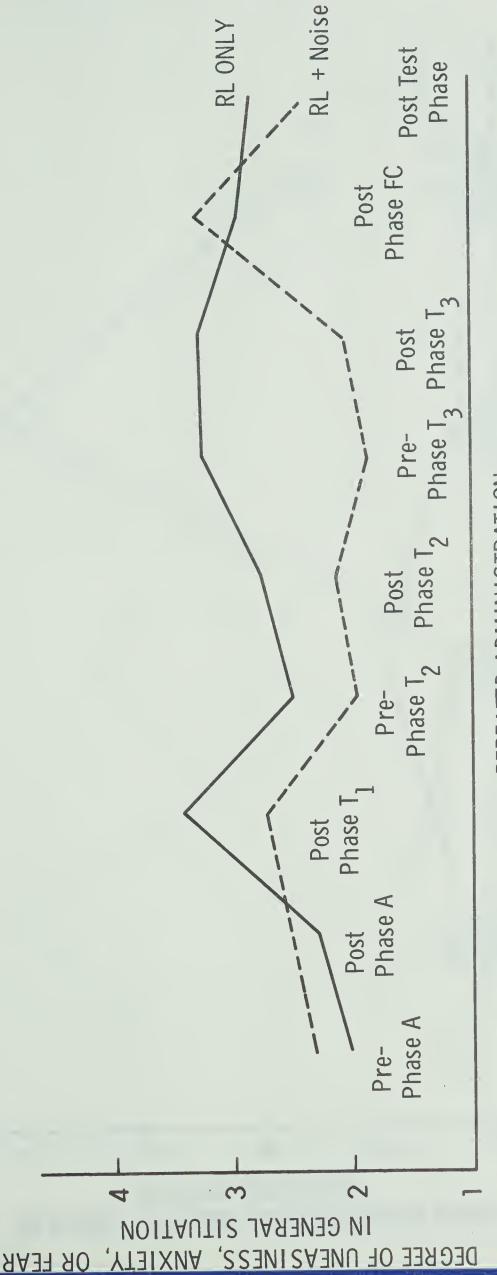


during phase FC (\underline{F} = 3.74, \underline{df} = 8/432, \underline{p} < .01, Figure 3). Their differences while not significant, except prior to phase T₃ (Tukey (a), \underline{q} = .98, \underline{p} < .01), are difficult to explain. No differences would have been expected until after phase FC.

Item 6: Degree of Uneasiness, Anxiety or Fear associated with RL: This item administered before and after phase A, and then after phase FC and the test phase in order to determine how successful RL + N was in conditioning fear. The FC X RA interaction was significant (\underline{F} = 17.75, \underline{df} = 3/162, \underline{p} < .01, Figure 4). The two groups (RL_{only} and RL + N) were comparable for the first two administrations of the item. For the last two administrations, RL + N \underline{S} s expressed significantly more anxiety associated with RL than the RL_{only} \underline{S} s. RL_{only} \underline{S} s maintained a consistently low level of anxiety to the RL. RL + N \underline{S} s expressed their greatest fear following phase FC but, following the test phase, their level of anxiety was still significantly higher than RL_{only} \underline{S} s (Tukey (a), \underline{q} = .98, \underline{p} < .01).

Item 7: Degree of Comfort: The interaction between Gs and RA was significant (\underline{F} = 3.01, \underline{df} = 8/432, \underline{p} < .01, Figure 5). Except for the last two administrations of ESQ, RL + N \underline{S} s reported more general comfort in the experimental situation. The administrations after phase A, before and after phase T3, were all significant (Tukey (a), \underline{q} = 1.04, \underline{p} < .05). The only occasion where RL + N Ss were lower was after they received white noise.

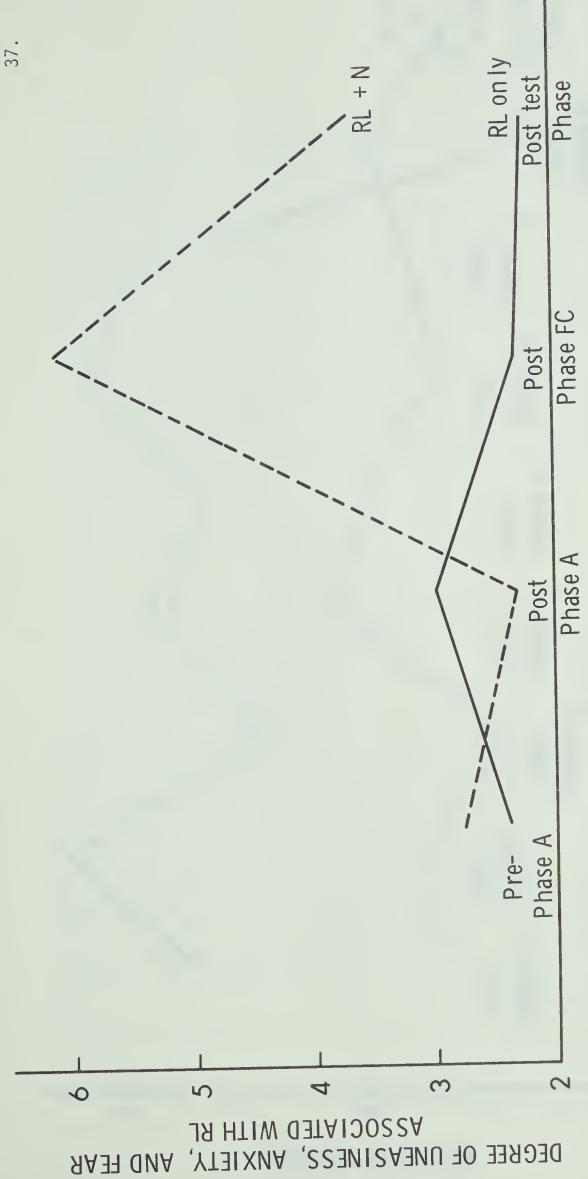




REPEATED ADMINISTRATION

Mean responses of RL only and RL + N Ss to ESQ Item 5: Degree of Uneasiness, Anxiety, or Fear in the General Situation as a function of repeated administration. FIGURE 3:

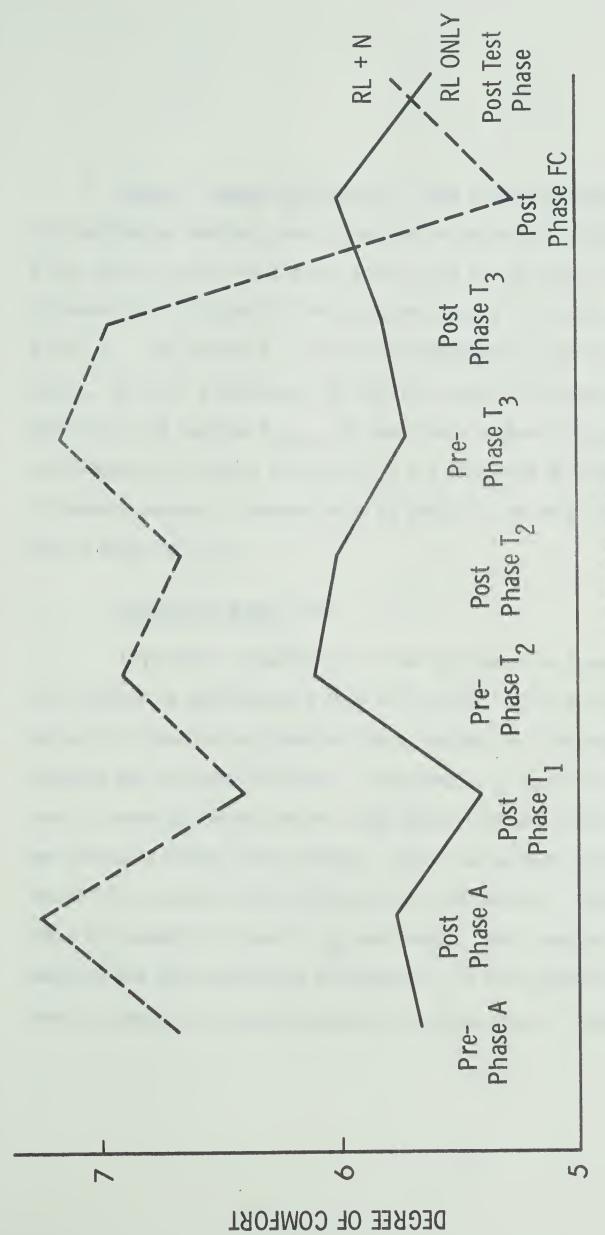




REPEATED ADMINISTRATION

Degree of Uneasiness, Anxiety, or Fear Associated with Mean responses of RL only and RL+N Ss to ESQ Item 6: RL as a function of repeated administration. FIGURE 4:





REPEATED ADMINISTRATION

Degree of Comfort as a function of repeated administration. FIGURE 5: Mean reponse of RL only and RL+N Ss to ESQ Item 7:



Item 8: Degree of Fatigue: Since a physiological variable was being recorded, any difference in degree of fatigue might alter this variable, Gs did not differ, but the FC groups were differentially affected as the study progressed ($\underline{F} = 2.62$, $\underline{df} = 8/432$, \underline{p} .01, Figure 6). Until the beginning of the last session, RL only and RL + N expressed the similar levels of fatigue. Throughout the last session RL only \underline{S} s reported a higher, but still moderate degree of fatigue (Tukey (a), $\underline{q} = 1.09$), than RL + N \underline{S} s. The difference between FC groups prior to phase FC can only be considered a sampling error.

Subjective Report (SR)

The unique dispositions of some $\underline{S}s$ cannot be accounted for by an objective questionnaire such as the ESQ. While one $\underline{S}'s$ response to an item may be identical with another, the reasons for the response may be quite different. For example, $\underline{S}s$ may vary in the level of meaning associated with the lights (red and green), which may adversely effect these results. Also, the methods reported by the HRT $\underline{S}s$ to control their HTs would be informative. After each phase and segment of phase T, $\underline{S}s$ were asked, "What thoughts or feelings did you have during this phase?" If this question did not evoke an answer, the questions were more structured: "Generally",



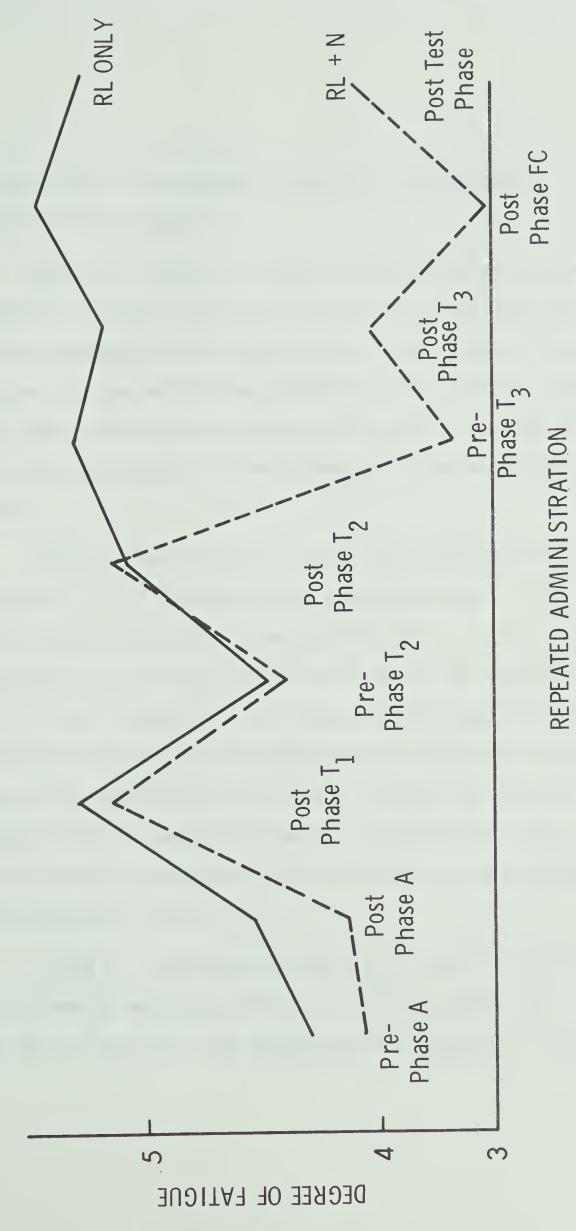


FIGURE 6: Mean responses of RL and RL+N Ss to ESQ Item 8: Degree of Fatigue as a function of repeated administration.



"in relationship to the phase", and "any particular meaning associated with the light."

<u>Phase A:</u> During this phase, all \underline{S} s experienced the same conditions. The usual responses to the red and green lights were that they reminded \underline{S} s of Christmas trees and stop lights. Several \underline{S} s stated that the red light was associated with alertness, whereas the green light was associated with relaxation. Although \underline{S} s expressed curiosity about the experiment, there was no suggestion of fear.

Phase <u>T</u>: Each of the three groups reacted differently to this phase. HRT <u>S</u>s' response to the tasks were varied. Since many HRT <u>S</u>s did not believe it was possible to control HR or, that they could do it, there was much frustration. RT <u>S</u>s reported discomfort in their right arm. They wished the GL to be on so they did not have to clench their fist any longer. When the GL appeared, there was considerable relief for the <u>S</u>s. Control <u>S</u>s reported boredom. They were anxious for the GL on to terminate. Since their task was counting how many times the GL occurred, they would become frustrated by the length of GL_{On} .

Phase FC: Ss who experienced RL_{only} found this phase quite boring. Some Ss reported difficulty in remaining awake. The other half of the Ss (RL + N) reported much displeasure. "I sat on



the edge of the chair waiting for the red light to come on.... I could never relax.": "That was terrible!"; "It was like a horror house."; "I was scared to death!"; and, "Where did you find such a terrible noise?" RL + N S had no difficulty associating the noise with RL_{on} .

<u>Test Phase</u>: Regardless of the group, <u>S</u>s reported that the occurrence of the RL interfered with the ongoing task. Control <u>S</u>s stated the most interference whereas HRT <u>S</u>s stated the least. By the last of the experiment, <u>S</u>s had little to say about the lights. By the conclusion of the test phase, no <u>S</u> reported fear of the RL.

With regard to the methods used to control HR, a few $\underline{S}s$ used respiration changes to vary HR, but most $\underline{S}s$ tried to think relaxing and anxious thoughts in accord with the task. One \underline{S} thought of relaxing in a hot bath when GL was present, and she thought of an engagement ring she had just recently received when the GL was absent. Another \underline{S} thought of a mountain scene to decrease her heart rate. She thought of all the tasks she had not completed and should have to increase her HR. Other $\underline{S}s$ were less specific in their response, saying simply that they thought either relaxing or exciting things. To increase her HR, one $\underline{S}s$ simply said "damn it, beat!" Most $\underline{S}s$ in this group felt that concentration was extremely important.



CHAPTER IV

DISCUSSION

Wolpe (1958) considers that anxiety is due mainly to a "widespread discharge of the autonomic nervous system, and predominantly of its sympathetic division." If fear is an autonomic response (sympathetic), then training a group of <u>S</u>s to decrease HR (parasympathetic) should inhibit the fear response. This statement is essentially Wolpe's principal of reciprocal inhibition. However, Wolpe uses muscular relaxation rather than autonomic control to inhibit fear in his procedure of systemic desensitization. The present study represents an experimental attempt utilizing recent physiological feedback methods to facilitate control of one autonomic response, heart rate. The basic assumption of the investigation is that if such autonomic control is possible it would be a more effective way of diminishing fear than through muscular relaxation.

Prior to the training phase, all groups of <u>S</u>s were comparable with respect to their HR and their responses to the first two administrations of ESQ. During the adaptation phase there was no evidence of HR differences between groups. Generally the HRs were between 80 and 90 bpm which was somewhat higher than any of the following phases. <u>S</u>s reported curiosity, but no uneasiness.



During the training phase the three groups differed in their response to the green light. Data indicated that both HRT and RT \underline{S} s produced a reduction in heart rate with GL_{on} , while control \underline{S} s went in the opposite direction. Although the HRT \underline{S} s consistently exhibited greater decreases in heart rate than the RT \underline{S} s, the differences between these groups were not statistically significant. However, these differences were significant during the test phase.

Most HRT <u>S</u>s exhibited heart rate changes of less than two beats per minute. One reason the differences were not large might have been the nature of feedback used. The meter fluctuated only one unit change for HR change of three beats. HRT <u>S</u>s were producing changes which were not markedly displayed to reward their efforts, even though they were succeeding at the task. Many HRT <u>S</u>s found the situation was extremely frustrating.

Brener, Kleinman, and Goesling (1969) found that <u>S</u>s could control their heart rate when told to do so. Without the aid of any external feedback they could both increase and decrease their HR. In the present experiment, several HRT <u>S</u>s performed progressively poorer as training continued. This decrease in performance might have been due to the aversive properties of the meter. In the first part of the training phase, HRT <u>S</u>s might have been depending more on their own "feedback" than that of the meter. As they relied more on the meter, the lack of marked change might have become punishing. These <u>S</u>s might have produced greater HR differences if they had continued to use their own cues.



In a study by Ascough and Sipprelle (1968), spontaneous increases and decreases of HR were controlled by verbal reinforcement. Verbal reinforcement - "That's good" - occurred one or two times per minute for marked increases or decreases in HR. In their experiment, over the five consecutive days of 31 minute sessions each, Ss demonstrated progressively larger HR changes. This gradual improvement was not in evidence in the present study. Seemingly, the present feedback method was not facilitating the task. This would suggest that feedback can facilitate HR changes although the particular feedback method used in this experiment did not.

Having the subject continuously monitor his HR is apparently not as effective as providing the subject with discrete information about a particular interbeat interval in the form of a verbal reinforcement (Ascough and Sipprelle, 1968). The periodic verbal feedback was based on changes the subject made in the desired direction. The present study's use of a monitor meant that each \underline{S} was receiving feedback whether it was a desired HR change or not. This study might have produced greater HR changes if feedback had been contingent on the desired change. In addition, the magnitude of reward in the present study was probably less than that produced by discrete verbal reinforcement delivered by an experimenter. In the present study HRT \underline{S} s presumably provided their own reward when the meter moved in the desired direction.



The fear conditioning phase failed to induce HR increases for RL + N Ss. During conditioning, the white noise was associated with a decrease in heart rate. Nevertheless, these S's reported a significant increase in fear. Taken at face value, the present data suggest that fear was associated with a decrease in HR. Wilson (1969), also, reported heart rate deceleration in response to a US of 83db. which sounded like a buzzing rasp. The CR was decelerative. Both Wilson and the present study used US of one second duration. De Leon (1964) used a 102 db. buzzer as a US to produce a decelerative UR. However, the CR was accelerative. De Leon's US was of a six second duration. In the present study, it is possible that the HR decrease was due to orienting behavior evoked by the white noise (Lacey, 1959). In the present experiment, the heart rate in response to the red light was recorded for a 7.5 second interval which included the occurrence of the white noise. Since the heart rate decrease was greater on trials with noise than on trials without noise, it is likely that this decrease was due to an unconditioned response to the noise rather than a conditioned response to the red light. It must be concluded that these data provide no evidence that any conditioning took place.

During the test phase there was no evidence of fear conditioning in the heart rate data. For all <u>Ss</u> there was a significant reduction in HR during the red light, whether or not it had been paired with white noise. However, the effect of the red



light did interact with the effect of the green light. HRT <u>Ss</u> exhibited their greatest decrease in HR when both the green and red lights were on (Figure 1). RT <u>Ss</u>, also exhibited this effect, but it was not as pronounced. Control <u>Ss</u> showed a slightly increased HR under these conditions. The augmenting effect of RL can not be due to fear, since this effect occurred for both the RL_{only} and RL + N groups. Instead, the decreased heart rate during RL might have been an orienting response (Lacey, 1959). When RL and GL occurred together, it is possible that the effect of the training that HRT and RT <u>Ss</u> received, summated with the effect of an orienting response to produce an even larger HR decrement than produced by either alone.

It was expected that HRT or RT $\underline{S}s$ would report less fear of the red light than the C $\underline{S}s$. The RL + N $\underline{S}s$, at the conclusion of the experiment, still reported a level of anxiety to the red light that was significantly higher than RL_{only} $\underline{S}s$, regardless of the type of training employed. There seem to be several possible reasons for the failure of HRT or RT $\underline{S}s$ to reduce fear. Several HRT $\underline{S}s$, who were able to vary their heart rates more than several beats, reported the use of thoughts to mediate HR changes. For example, one \underline{S} thought of "a warm bath" to decrease heart rate and thought of "all tasks I didn't complete today", to increase heart rate. Much concentration was reported. Successful HRT

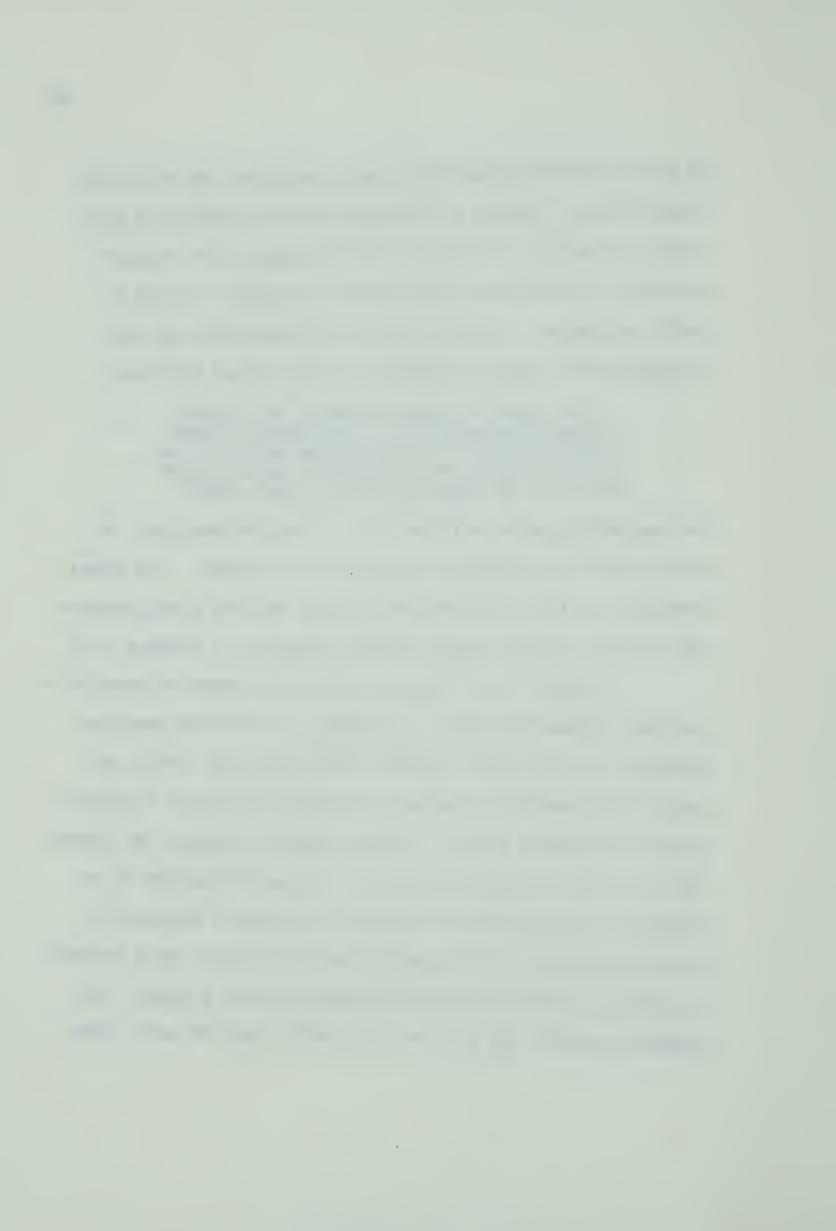


<u>S</u>s did not perform as well when they reported they had difficulty concentrating. Inducing a HR decrease does not necessarily mean "mental relaxation." As Rachman (1967) suggests, the important variable in relaxation might be "mental relaxation" instead of bodily relaxation. Actually the use of concentration may have interfered with "mental relaxation", as Elmer Green describes:

...You visualize what you want to have happen, instruct the body to do it, and then you detach yourself from it. That's passive volition. Use active volition and the autonomic nervous system does just the opposite, usually (Green, 1969).

For many HRT <u>Ss</u> active volition (i.e., trying too hard) may, in part, explain why larger HR changes were not obtained. The arousal produced by active concentration in HRT <u>Ss</u> may have offset whatever fear reducing effects might have been produced by a decrease in HR.

In order to be effective, perhaps the control of muscular or autonomic relaxation should be automatic, not requiring conscious mediation. Wolpe (1958), Jacobson (1938), and Yates (1947), emphasize the necessity of having the response of muscular relaxation under the patient's control. Through repeated practice, the patient learns to induce relaxation quickly. This quick induction of relaxation is probably just as essential to autonomic responses as muscular responses. Schultz and Luthe (1959) require their patients to practice associating relaxing phrases with bodily states. For example, a patient who was practicing control over HR would, after



inducing a state of passive volition and relaxation, say to himself a phrase suggesting that his heart rate was calm and regular. Progressively, the phrase becomes associated with the bodily state. The patient can ultimately evoke the state at will and distinguish it from other heart rate states. It is not clear how the phrase becomes associated with the HR state, but it appears similar to Yates' (1947) method of associating muscular relaxation to a word that could later evoke relaxation. A patient lies down on a couch and relaxes. If he falls asleep, it is quite acceptable. As he is relaxing, he repeats the word "calm". Over a period of time, the word "calm" evokes the state of relaxation. Once the association is strong enough for the word to evoke the relaxation, the patient can "set" himself to relax on any occasion he chooses.

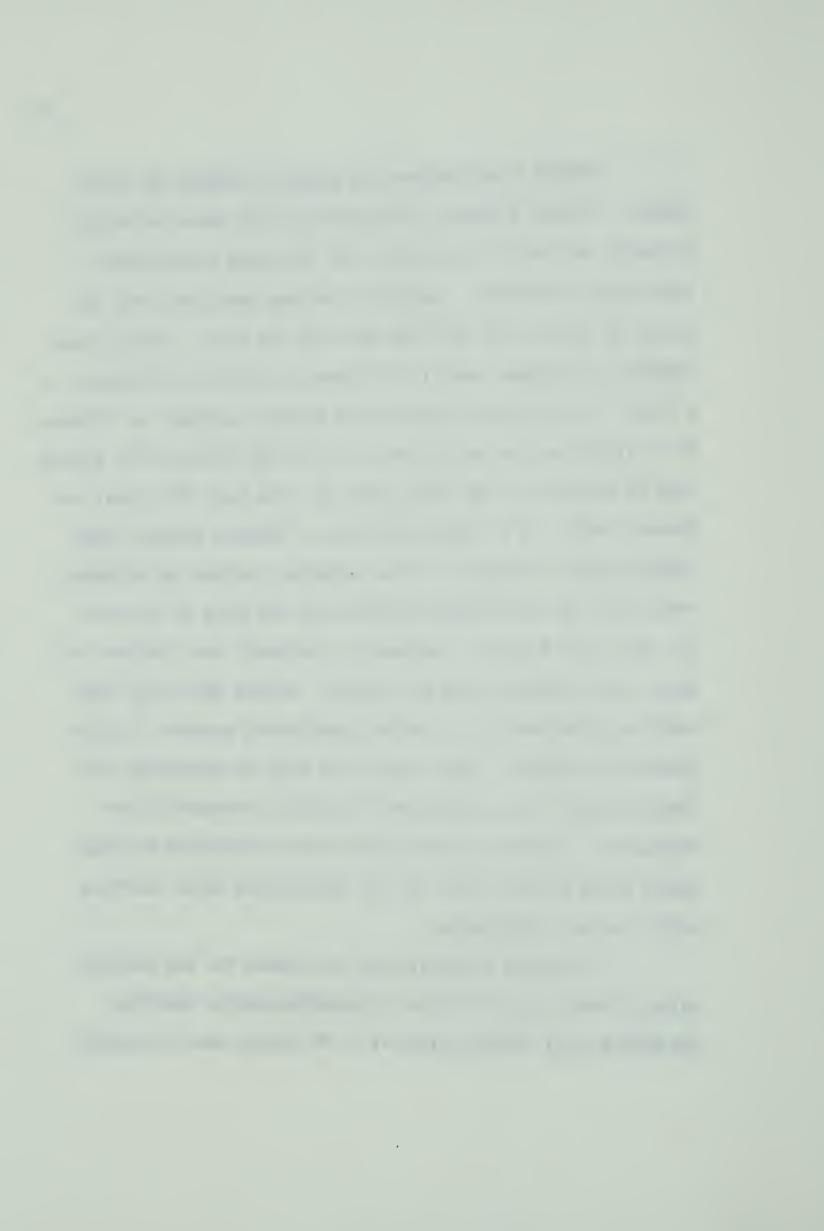
In order to train <u>Ss</u> in effective HR control it might be necessary to classically condition the HR change. An efficient way to attain this control might be to initially produce the HR change by instrumental training, and subsequently classically condition this change. Utilizing Ascough and Sipprelle's (1968) method for reinforcing HR decrease, the achieved decrease might be paired to a stimulus, such as the word "calm". This word might then evoke a state of HR slowing. That is, under these conditions the HR decrease might not require active mediation, and might possibly be fear reducing.



Another issue concerns the amount of feedback Ss should receive. Brener, Kleinman, and Goesling (1969) demonstrated that by simply telling Ss to vary their HR, that they could produce significant HR changes. Seemingly knowledge about what they are trying to control does not interfere with the task. However, when feedback is provided, what is its effect on the desired results? In a study in which Ss were receiving HR feedback but were not informed of HR slowing as the task, four out of five Ss who correctly guessed that HR decrease was the task, could not slow their HRs (Engel and Hansen, 1966). At a recent conference of feedback methods, Engel (1969) stated "I've been a little concerned, without any evidence, that if you tell the subject too much you are going to screw him up. But I don't know." Just what is "too much" and "too much" of what, the conference could not resolve. Perhaps telling Ss "too much" may interfere with classical conditioning because it evokes cognitive processes. These congnitions might be associated with bodily states, i.e., tension, which would be incompatible with relaxation. Therefore, Ss who have no more information than necessary might be less likely to have associations which interfere with classical conditioning.

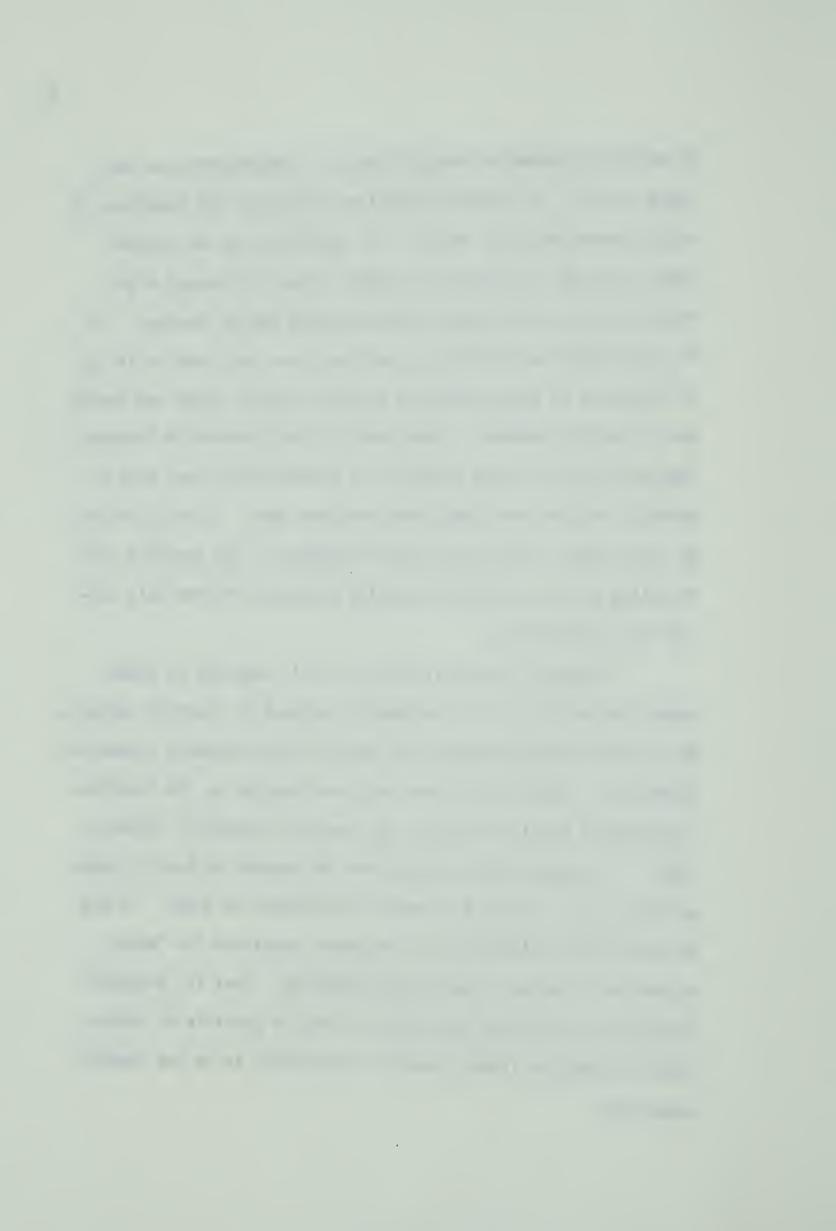
The present study provided no evidence for the possible value of heart rate training as a psychotherapeutic technique.

The method might be more effective if HR control could utlimately



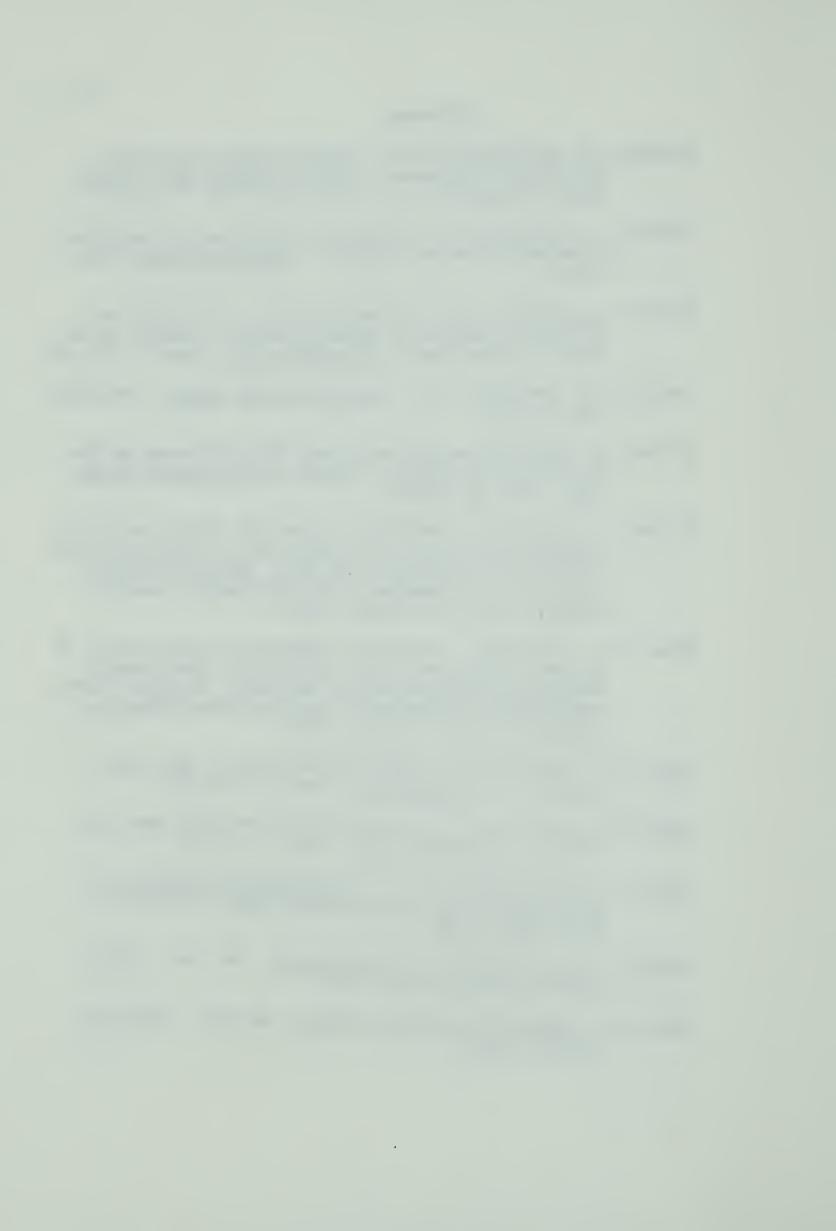
be made more automatic through classical conditioning, as indicated earlier. An important question relates to the magnitude of reinforcement which <u>Ss</u> receive. In comparison to the present study, Ascough and Sipprelle's (1968) larger HR changes might have been due to the greater reinforcement the <u>Ss</u> received. As Di Cara (1969) has suggested, there may be a real need to aid <u>Ss</u> in attending to the appropriate internal signal, above and beyond just providing feedback. Even though <u>Ss</u> may receive HR feedback, they may have paid more attention to other bodily cues, such as skeletal muscles, which may have confused them. It could be that <u>Ss</u> first need a simple, but basic knowledge of the possible control they can have over their bodily processes and how this control can benefit them.

Research in several areas is still required to understand the possible uses of autonomic feedback in treating neurosis. More needs to be known about the muscular and autonomic relaxation technique. Specifically, more data are required on the relationship between bodily relaxation and "mental relaxation" (Rachman, 1967). It appears that a person must be capable of bodily states of relaxation in order for "mental relaxation" to occur. It may be that bodily relaxation is a necessary condition for "mental relaxation", but not a sufficient condition. That is, although the two may usually be associated, it may be possible to induce bodily relaxation without "mental relaxation", as in the present experiment.

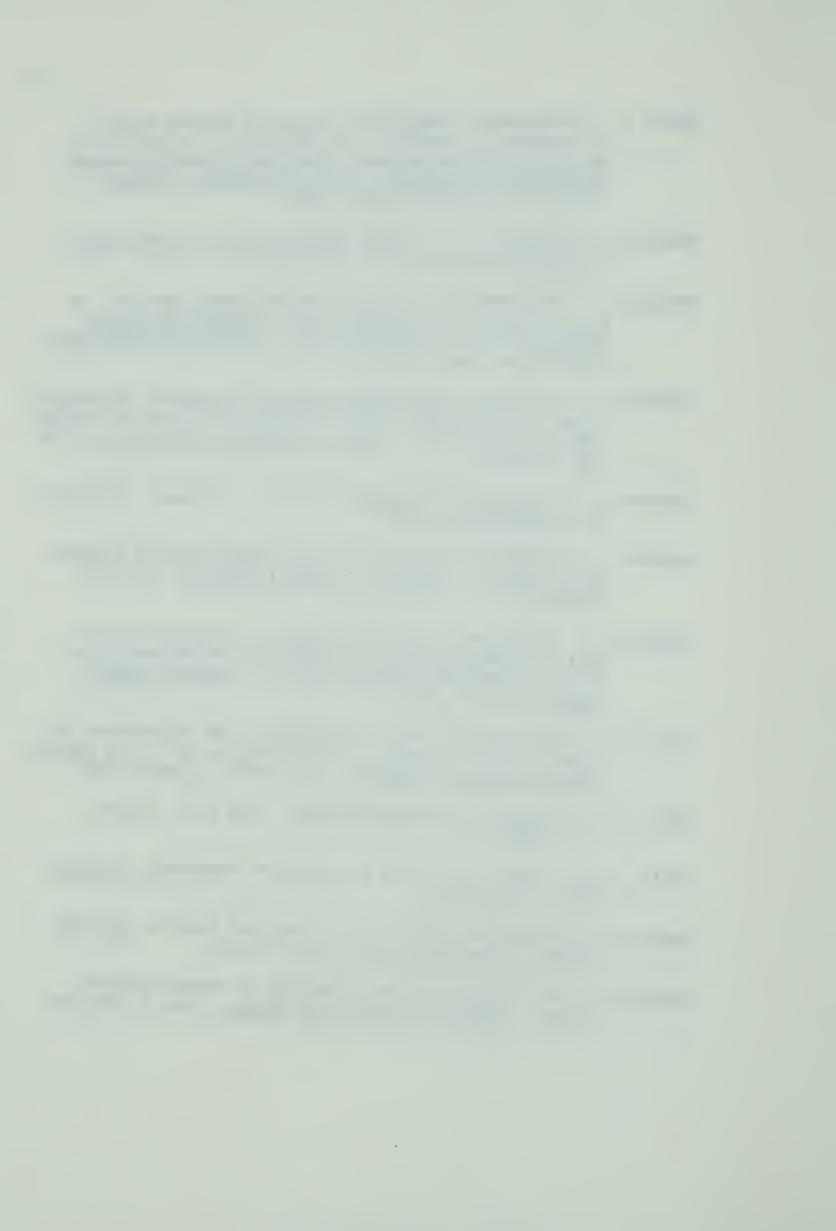


References

- Ascough, J.C. and Sipprelle, C.N. Operant verbal conditioning of autonomic responses. Behavior Research and Therapy, 1968, 6, 363-370.
- Brener, J. and Hotersall, D. Heart rate control under conditions of augmented sensory feedback. <u>Psychophysiology</u>, 1966, 3, 28-32.
- Brener, J., Kleinman, R.A., and Goesling, W.J. The effects of different exposures to augmented sensory feedback on the control of heart rate. <u>Psychophysiology</u>, 1969, 5, 510-516.
- Cattell, R.B. and Scheir, I.H. <u>Neuroticism</u> and <u>anxiety</u>. New York: Ronald, 1961.
- De Leon, G. Conditioning the human heart rate with noise as the unconditioned stimulus. <u>Journal of Experimental Psychology</u>, 1964, 68, 518-520.
- Di Cara, L. (Panelist). Mechanisms underlying learned control of autonomic functions. In W. Fenz (Chm.), Conditioning and the control of autonomic functions. Research reports presented at the Meeting of the Bio-Feedback Research Society, Palo Alto, October, 1969.
- Engel, B.T. (Panelist). Mechanisms underlying learned control of autonomic functions. In W. Fenz (Chm.), Conditioning and the control of autonomic functions. Research reports presented at the Meeting of the Bio-Feedback Research Society, Palo Alto, October, 1969.
- Engel, B.T. and Chism, R. Operant conditioning of heart rate speeding. Psychophysiology, 1967, 3, 418-426.
- Engel, B.T. and Hansen, S.P. Operant conditioning of heart rate slowing. <u>Psychophysiology</u>, 1966, 3, 176-187.
- English, H.B. and English, A.C. <u>A comprehensive dictionary of psychological and psychoanalytical terms</u>. New York: David McKay, 1958.
- Eysenck, H.J. <u>The effects of psychotherapy</u>. New York: International Science Press, 1966.
- Fink, D.H. Release from nervous tension. New York: Simon and Schuster, 1943.



- Green, E. (Discussant). Mechanisms underlying learned control of autonomic functions. In W. Fenz (Chm.), Conditioning and the control of autonomic functions. Research reports presented at the Meeting of the Bio-Feedback Research Society, Palo Alto, October, 1969.
- Hnatiow, M. and Lang, P.J. Learned stabilization of cardiac rate. Psychophysiology, 1965, 1, 330-336.
- Krasner, L. The therapist as a social reinforcement machine. In H.H. Strupp and L. Luborsky (Eds.), Research in psychotherapy, Vol. II, Washington, D.C.: American Psychological Association, 1962, 61-94.
- Jacobson, E. Electrical measurements concerning muscular contraction (Tonus) and the cultivation of relaxation in man-relaxation-times of individuals. American Journal of Physiology, 1934, 108, 573-580.
- Jacobson, E. <u>Progressive relaxation</u> (2nd Ed.). Chicago: University of Chicago Press, 1938.
- Jacobson, E. Variation of pulse rate with skeletal muscle tension and relaxation. Annals of Internal Medicine, 1940, 13, 1619-1625.
- Jacobson, E. Relaxation training of flyers in the United States
 Navy preliminary report on Neufeld's "Brief evaluation
 of the relaxation training program." American Psychologist, 1947, 2, 424.
- Lacey, J.I. Interpolated remark in Grastyan's, the hippocampus and higher nervous activity. In M.A. Brazier (Ed.), <u>The central nervous system and behavior</u>. New York: J. Macy, 1959.
- Maher, B.A. <u>Principles of psychopathology</u>. New York: McGraw-Hill, 1966.
- Miller, N.E. Learning of visual and glandular responses. Science, 1969, 163, 434-445.
- Neufeld, W. Relaxation methods in U.S. Navy Air Schools, American Journal of Psychiatry, 1951, 108, 132-137.
- Rachman, S. The role of muscular relaxation in desensitization therapy. Behavior Research and Therapy, 1968, 6, 159-166.



- Ruesch, J. and Finesinger, J.E. The relationship between electromyographic measurements and subjective reports of muscular relaxation. Psychosomatic Medicine, 1943, 5, 132-138.
- Schultz, H.J. and Luthe, W. <u>Autogenic training</u>: <u>A psychophysiologic approach in psychotherapy</u>. New York: Greene and Stratten, 1959.
- Shearn, D.W. Operant conditioning of heart rate. Science, 1962, 137, 530-531.
- Sherrington, C.S. The integrative action of the nervous system.

 New Haven, Conn.: Yale University Press, 1906 (Reprinted, 1961).
- Strupp, H.H. Psychotherapeutic technique, professional affiliation, and experience level. <u>Journal of consulting psychology</u>, 1955, 19, 197-202.
- Ullman, L.P. and Krasner, L. (Eds.). <u>Case studies in behavior modification</u>. New York: Holt, 1965.
- Winer, B.J. <u>Statistical principles in experimental design</u>. New York: McGraw-Hill, 1962.
- Wolpe, J. <u>Psychotherapy by reciprocal inhibition</u>. Stanford: Stanford University Press, 1958.
- Yates, D.H. Relaxation in psychotherapy. <u>Journal of general psychology</u>, 1946, 34, 213-237.



APPENDIX A

SUBJECT PERMISSION SLIP

I recognize that this experiment involves a minimal possibility of risk. I am willing to accept total responsibility for my participation. Therefore, I will not hold the examiner or the University of Alberta responsible.

date		name	
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APPENDIX B

Items From Experimental Situation Questionnaire.*

				((circle one)			
1.	Degree of Interest.	1 Low	2	3	4 5 6 Moderate	7	8	9 H i gh
2.	Degree of Restlessness.	1 Low	2	3	4 5 6 Moderate	7	8	9 High
3.	Degree of Uneasiness, Anxiety, or Fear associated with GLon.	1 Low			4 5 6 Moderate	7	8	9 H i gh
4.	Degree of Uneasiness, Anxiety, or Fear associated with GL _{off} .	1 Low		3	4 5 6 Moderate	7	8	9 H i gh
5.	Degree of Uneasiness, Anxiety, or Fear in the General Situation.			3	4 5 6 Moderate	7	8	9 H i gh
6.	Degree of Uneasiness, Anxiety, or Fear associated with RL.	l Low		3	4 5 6 Moderate	7	8	9 High
7.	Degree of Comfort.	l Low	2	3	4 5 6 Moderate	7	8	9 High
8.	Degree of Fatigue.] Low	2	3	4 5 6 Moderate	7	8	9 High

^{*} Items 1,2,5,7, and 8 were repeated on every administration of ESQ. Items 3 and 4 were not administrated after phase FC. Item 6 was administrated before and after the adaptation phase and the test phase.



APPENDIX C

ANALYSIS OF VARIANCE TABLES FOR HEART RATE



Analysis of Variance For Heart Rate During the Adaptation Phase

Source	DF	MS	F
Groups (G)	2	.98	.27
Fear Conditions (FC)	1	112.23	3.05
Trials (T)	2	13.44	2.86
Periods (P)	7	1.29	1.64
G x FC	2	39.01	1.06
FC x T	4	3.99	0.85
GxT	2	.54	0.11
CxP	14	. 46	0.59
GxP	7	.68	0.87
TxP	14	.35	0.52
Subject x (CG)	54	36.80	
$C \times G \times T$	4	1.49	0.32
CxGxP	14	.48	0.61
$C \times T \times P$	28	.45	0.67
GxTxP	14	1.00	1.49
S x T (CG)	108	4.70	
S x P (CG)	378	.78	
CxGxTxP	28	. 77	1.15
S x T x P (CG)	756	.67	



Analysis of Variance For Heart Rate During Phase T_1

Source	DF	MS	F
Groups (G)	2	52.50	.20
Trials (T)	19	16.01	1.21
Green Light (GL)	1	57.82	14.32*
Periods (P)	3	1.09	1.46
Subjects (G)	57	263.37	
GxT	38	14.02	1.06
G x GL	2	23.55	5.83*
T x GL	19 6 57	.63	0.78
GxP	6	1.13	1.52
TxP	57	.51	0.94
GL x P	3	1.36	1.24
S x T (G)	1083	13.19	
S x GL (G)	57	4.04	
SxP(G)	171	.74	
GxTxL	38	.97	1.20
GxTxP	114	.74	1.38
G x GL x P	6	.91	0.83
TxGLxP	57	.62	1.14
$S \times T \times GL(G)$	1083	.81	
S x T x P (G)	3249	.54	
S x GL x P (G)	171	1.10	
GXTXGLXP	114	.67	1.23
$S \times T \times GL \times P (G)$	3249	. 54	



Analysis of Variance for Heart Rate During Phase T_2

Source	DF	MS	F
Groups (G)	2	381.15	1.79
Trials (T)	19	5.17	0.84
Green Light (GL)	1	24.30	12.12*
Periods (P)	3	1.23	1.51
Subjects (G)	57	212.91	
GxT	38	3.06	0.50
G x GL	2	25.99	12.97*
T x GL	19	1.48	1.62
GxP	6	.78	0.96
TxP	57	.47	0.90
GL x P	3	1.86	1.90
$S \times T (G)$	1083	6.12	
S x GL (G)	57	2.00	
SxPxG	171	.81	
G x T x GL	38	1.46	1.60
GxTxP	114	.54	1.01
G x GL x P	6	.76	0.78
T x GL x P	57	.61	1.08
$S \times T \times GL (G)$	1083	.91	
$S \times T \times P (G)$	3249	.53	
$S \times GL \times P(G)$	171	.98	
GXTXGLXP	114	.74	1.29
$S \times T \times GL \times P(G)$	3249	.57	



Source	<u>DF</u>	MS	<u>F</u>
Groups (G) Trials (T)	2 9 1 3	715.98 55.22	3.47 4.16*
Green Light (GL) Periods (P)		3.00 1.03	1.39 1.21
Subjects (G) G x T	57 18	206.23 58.76	4.42*
G x GL T x GL	2 9 6	10.57 6.59	4.91 2.36
G x P T x P	27	2.94	3.47* 1.35
GL x P S x T (G)	3 513	2.23 13.29	1.65
S x GL (G) S x P (G)	57 171	2.15 .85	2 52+
G x T x GL G x T x P	18 54	9.84 1.62	3.52* 2.03*
G x GL x P T x GL x P	6 27	.43	0.32
S x T x GL (G) S x T x P (G)	513 1539	2.79	
S x GL x P (G) G x T x GL x P	171 54	1.35 1.25	1.57*
$S \times T \times GL \times P (G)$	1539	.80	



Analysis of Variance for Heart Rate During the Fear Conditioning Phase

DF	MS	F
2	197.66	4.04
1	47.88	0.98
19	1.43	1.34
1	3.45	1.84
2	145.29	2.97
38	1.50	1.41
19	.90	0.84
2	.35	0.19
1	18.90	10.10*
19	.55	0.82
54	48.88	
38	1.32	1.23
2	3.50	1.87
38	.59	0.89
		1.80
54	1.87	
38	.54	0.82
1026	.67	
	2 1 19 1 2 38 19 2 1 19 54 38 2 38 19 1026 54	2 197.66 1 47.88 19 1.43 1 3.45 2 145.29 38 1.50 19 .90 2 .35 1 18.90 19 .55 54 48.88 38 1.32 2 3.50 38 59 19 1.20 1026 1.07 54 1.87 38 .54



Analysis of Variance of Noise and No Noise Trials for RL + N \underline{S} s During Phase FC

Source	DF	MS	<u>F</u>
Groups (G)	2	13.77	0.25
Noise (N)	1	.27	0.19
Trials (T)	9	2.04	1.38
Periods (P)		19.25	6.51
Subjects (G)	27	54.55	3 40
G x N	2	2.03	1.42
G x T	18	.80	0.54
N x T	9	1.34	1.36
G x P N x P	9 2 1	.85	0.29 8.22*
	9	8.67 .50	0.66
T x P S x N (G)	27	1.43	0.00
S x T (G)	243	1.48	
S x P (G)	27	2.96	
GXNXT	18	2.01	2.04*
GXNXP	2	2.35	2.23
GXTXP	18	.43	0.56
NxTxP	9	.92	1.21
$S \times N \times T (G)$	243	.99	
$S \times N \times P (G)$	27	1.05	
$S \times T \times P (G)$	243	.76	
GXNXTXP	18	.38	0.50
$S \times N \times T \times P (G)$	243	.76	



Analysis of Variance for Heart Rate During the First Analysis of the Test Phase

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC) Green Light (GL) Trials (T) Periods (P) G x FC G x GL FC x GL G x T FC x T GL x T GL x T G x P FC x P Subjects (G x FC)	2 1 1 9 3 2 2 1 18 9 9 6 3 3 27 54	102.55 86.67 7.92 .70 3.68 335.65 15.69 2.04 1.39 1.78 1.10 .96 .12 .38 .46 91.37	1.12 0.95 4.76 0.44 5.01* 3.67 9.44* 1.23 0.87 1.11 1.39 1.31 0.17 0.59 0.94
G x FC x GL G x FC x T G x GL x T FC x GL x T G x FC x P G x GL x P FC x GL x P FC x GL x P FC x GL x P S x GL (G x FC) S x T (G x FC) S x P (G x FC)	2 18 18 9 6 6 3 54 27 27 54 486 162	1.77 1.20 1.12 1.69 2.04 .36 .35 .59 .58 1.66 1.60	0.13 1.11 1.51 1.41 2.30 3.17* 0.55 0.72 1.20 1.19
G x FC x GL x T G x FC x GL x P G x FC x T x P G x GL x T x P FC x GL x T x P S x GL x T (G x FC) S x GL x P (G x FC) S x T x P (G x FC)	18 6 54 54 27 486 162 1458	.73 .79 .21 .59 .53 .30 .79 .64	1.00 0.32 1.20 1.09 0.61
G x FC x GL x T x P S x GL x T x P (G x FC)	54 1458	.46	0.94



Analysis of Variance for Heart Rate During the Second Analysis of the Test Phase

Source	DF	MS	F
Groups (G) Fear Condition (FC) Green Light (GL) Trials (T) Periods (P) G x FC G x GL FC x GL G x T FC x T GL x T GL x T G x P FC x P Subjects (G x FC)	2 1 1 4 3 2 2 1 8 4 4 6 3 3 12 54	84.38 82.14 .14 .72 3.83 192.22 1.44 1.93 .69 .72 1.05 .63 .13 .61 .65 46.24	1.82 1.78 0.08 0.54 7.75* 4.16 0.81 1.08 0.52 0.54 1.11 1.28 0.26 1.07 1.37
Subjects (G x FC) G x FC x GL G x FC x T G x GL x T FC x GL x T G x FC x P G x GL x P FC x GL x P FC x GL x P G x T x P FC x T x P GL x T x P S x GL (G x FC) S x T (G x FC)	2 8 8 4 6 6 3 24 12 12 54 216	.61 1.93 .85 .78 .18 1.07 .45 .40 .24 .39 1.78	0.34 1.45 0.90 0.82 0.36 1.89 0.79 0.83 0.50 0.82
S x P (G x FC) G x FC x GL x T G x FC x GL x P G x FC x T x P G x GL x T x P FC x GL x T x P S x GL x T (G x FC) S x GL x P (G x FC)	162 8 6 24 24 12 216 162	.49 .33 .74 .87 .62 .46 .94	0.35 1.30 1.84* 1.32 0.97
S x T x P (G x FC) G x FC x GL x T x P S x GL x T x P (G x FC)	648 24 648	. 48 . 42 . 47	0.88



APPENDIX D

ANALYSIS OF VARIANCE FOR EACH ITEM OF THE EXPERIMENTAL SITUATION QUESTIONNAIRE (ESQ)



Analysis of Variance for ESQ, Item #1:

Degree of Interest

Source	DF	MS	<u>F</u>
Groups (G)	2	107.58	3.19
Fear Condition (FC)		46.82	1.39
Repeated Administrations (RA)	8	7.91	4.21
G x FC	2	12.15	0.36
G x RA	16	2.24	1.19
FC x RA	8	3.35	1.78
Subjects (G x FC)	54	33.75	
G x FC x RA	16	1.27	0.67
S x P (G x GL)	432	1.88	

* <u>p</u> < .01

Analysis of Variance for ESQ, Item #2:

Degree of Restlessness

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC) Repeated Administration (RA) G x FC G x RA FC x RA Subjects (G x FC)	2 1 8 2 16 8 54	.90 6.45 24.83 18.04 2.81 4.05 16.75	0.05 0.38 11.59* 1.08 1.31 1.89
G x FC x RA	16	1.73	0.80
S x P (G x GL)	432	2.14	



Analysis of Variance for ESQ, Item #3:

Degree of Uneasiness, Anxiety, or Fear Associated with GLon

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC) Repeated Administration (RA) G x FC G x RA FC x RA Subjects (G x FC)	2 1 7 2 14 7 54	1.61 9.08 5.62 4.27 1.74 .42 8.07	0.20 1.12 5.78 * 0.53 1.79 0.43
G x FC x RA S x P (G x GL)	14 378	.74 .97	0.76

* p < .01

Analysis of Variance for ESQ, Item #4:

Degree of Uneasiness, Anxiety, or Fear Associated with GL_{off}

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC) Repeated Administration (RA) G x FC G x RA FC x RA Subjects (G x FC) G x FC x RA	2 1 7 2 14 7 54	53.65 14.01 18.45 16.76 5.84 .68 10.81 1.69	4.96 1.30 9.72* 1.55 3.08* 0.36
S x P (G x GL)	378	1.90	



Analysis of Variance for ESQ, Item #5:

Degree of Uneasiness, Anxiety, or Fear in the General Situation

Source	DF	MS	E
Groups (G) Fear Condition (FC)	2	25.72 25.79	1.45
Repeated Administration (RA)	8	7.18	4.57*
G x FC	2	1.61	0.09
G x RA	16	2.91	1.85
FC x RA	8	5.86	3.74*
Subjects (G x FC)	54	17.78	
G x FC x RA	16	.86	0.55
$S \times P (G \times GL)$	432	1.57	

* p < .01

Analysis of Variance for ESQ, Item #6:

Degree of Uneasiness, Fear, or Anxiety Associated with RL

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC)	2	5.72 93.75	0.85 13.91*
Repeated Administration (RA) G x FC	3 2	37.21 3.05	11.58*
G x RA FC x RA	6 3	2.56 57.03	0.80 17.75*
Subjects (G x FC) G x FC x RA S x P (G x GL)	54 6 162	6.74 .73 3.21	0.23



Analysis of Variance for ESQ, Item #7: Degree of Comfort

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC)	2	51.96 50.42	2.97 2.88
Repeated Administration (RA) G x FC	8 2	7.39 22.54	2.74* 1.29
G x RA	16	2.57	0.95
FC x RA Subjec t s (G x FC)	8 54	8.11 17.49	3.01*
G x FC x RA	16	3.52	1.31
S x P (G x GL)	432	2.70	

 $\star p < .01$

Analysis of Variance for ESQ, Item #8: Degree of Fatigue

Source	DF	MS	<u>F</u>
Groups (G) Fear Condition (FC)	2	9.18 71.87	0.46
Repeated Administration (RA)	8	6.93	2.27
G x FC	2	21.90	1.10
G x RA	16	2.85	0.94
FC x RA	8	8.01	2.62*
Subjects (G x FC)	54	19.90	
G x FC x RA	16	1.83	0.60
$S \times P (G \times GL)$	432	3.05	



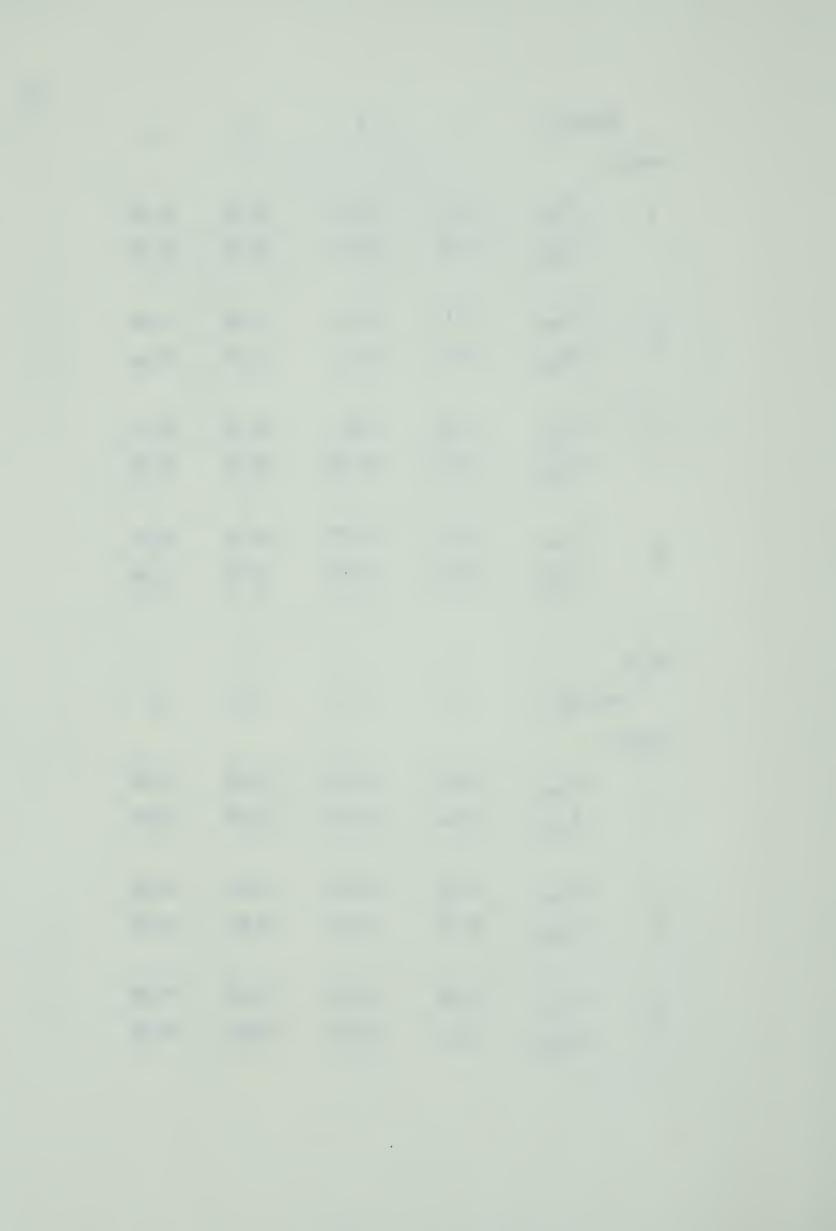
APPENDIX E - Phase T₃

Mean Heart Rate For The Three Experimental Groups As A Function Of $\operatorname{GL}_{\operatorname{on}}$ And $\operatorname{GL}_{\operatorname{off}}$, Periods, And Trials

HRT Ss					
	Periods	1	2	3	4
Trials					
1	GLon	10.00	10.05	9.90	10.10
1	GL _{off}	10.40	10.10	10.20	9.95
	GLon	10.30	10.20	10.05	10.35
2	GLoff	10.10	10.00	10.30	10.30
2	GLon	10.20	10.45	10.10	10.05
3	GL _{off}	10.10	10.10	10.40	10.30
4	GLon	10.20	10.00	10.15	10.00
4	GL _{off}	10.25	10.50	10.35	10.15
	GLon	10.10	10.25	10.15	10.35
5	GL _{off}	10.65	10.45	10.55	10.45
	GLon	9.95	9.95	10.15	10.00
6	GLoff	10.55	10.45	10.40	10.60



Periods		1	2	3	4
Trials					
7	GLon	10.10	9.85	10.00	10.30
/	GL _{off}	10.85	10.25	10.45	10.35
0	GLon	10.15	10.15	10.00	10.25
8	GLoff	10.35	10.00	10.55	10.45
9	GLon	10.00	9.80	10.15	10.15
9	GLoff	10.15	10.20	10.70	10.30
30	GLon	10.25	10.25	10.15	10.20
10	GLoff	10.30	10.40	10.10	10.30
RT <u>S</u> s					
Periods		1	2	3	4
Trials					
1	GLon	10.15	10.45	10.35	10.50
	GLoff	10.40	10.60	10.40	10.00
2	GLon	10.20	10.80	11.05	10.50
2	GLoff	10.35	10.20	10.60	10.25
2	GLon	10.05	10.75	10.35	10.90
3	GLoff	9.95	10.25	10.25	10.25



Trials					
4	GLon	10.30	10.60	10.40	10.20
4	GLoff	10.30	10.40	10.40	10.35
5	GLon	10.20	10.15	10.10	10.05
	GLoff	10.35	10.35	10.40	10.30
	GI	10.40	10.40	8.00	8.00
6	GL on	8.30	8.15	8.30	7.90
	GLoff	0.30	0.13	0.30	7.50
	GLon	8.15	8.55	8.15	8.05
7	GLoff	8.20	8.20	8.35	8.30
8	GLon	8.35	8.25	8.35	8.35
· ·	GLoff	8.20	8.40	8.40	8.00
9	GLon	8.05	8.35	8.30	8.10
	GLoff	8.05	8.25	8.30	8.10
	GLon	8.15	8.05	8.30	7.85
10	GL off	10.00	10.15	10.15	10.00
	011				
Control	<u>S</u> s				
Periods		1	2	3	4
Trials					
3	GLon	10.45	10.55	10.50	10.75
1	GLoff	10.85	10.40	10.50	10.65



Periods		1	2	3	4
Tri	als				
2	GLon	10.75	10.95	10.45	10.85
2	GL _{off}	10.80	10.75	10.80	10.55
3	GLon	10.70	11.05	10.95	10.85
3	GL _{off}	10.90	10.85	10.70	10.85
4	GLon	10.90	11.25	10.90	11.25
4	GL _{off}	10.60	10.75	10.75	10.65
5	GLon	10.70	11.00	10.85	10.95
5	GL _{off}	10.75	10.80	10.80	10.55
6	GLon	10.80	10.85	10.70	10.90
O	GLoff	10.45	10.85	10.85	10.95
7	GLon	10.90	10.85	11.00	10.85
,	GL _{off}	10.70	10.70	10.45	10.40
8	GLon	10.90	10.95	10.90	10.95
	GLoff	10.90	11.00	10.80	11.10
9	GLon	10.75	10.85	10.65	10.70
J	GL _{off}	10.65	10.80	10.75	10.90
10	GLon	10.80	10.70	10.85	10.80
10	GL _{off}	10.70	10.45	10.80	10.45



APPENDIX F

Mean Heart Rate For The Three Experimental Groups As A Function Of Fear Conditioning, Trials, And Periods HRT $\underline{S}s$

		1	Red Light only		
	Periods	1	2	3	4
Trials					
1 2 3 4 5		10.15 9.90 10.05 10.05 9.70	10.15 9.90 10.00 10.25 9.75	10.20 9.90 9.65 9.90 9.75	9.55 9.85 9.75 9.80 9.70
		I	Red Light + Noi	se	
	Periods	1	2	3	4
Trials					
1 2 3 4 5		10.00 10.65 10.55 10.20 10.30	10.50 10.35 10.45 10.15 10.50	10.35 10.05 10.25 10.10 10.30	10.15 10.05 10.30 10.10 10.00
RT Ss					
			Red Light only	,	
	Periods	1	2	3	4
Trials					
1 2 3 4 5		9.65 10.00 10.00 10.20 10.35	10.35 9.80 10.15 10.05 10.20	9.85 9.75 9.85 9.85 9.95	9.75 10.10 9.75 10.10 9.65



Red	liaht	+	Noisa

	Red Light + Noise					
	Periods	1	2	3	4	
Trial	S					
1 2 3 4 5		10.30 9,95 10.10 10.10 9.85	10.05 10.15 10.25 9.90 9.70	10.45 10.05 9.85 9.75 9.75	10.20 9.85 9.70 9.95 9.75	
C <u>S</u> s						
		Re	ed Light only	/		
	Periods	1	2	3	4	
Trial	S					
1 2 3 4 5		11.40 11.25 11.50 11.05 11.30	11.25 11.35 11.45 11.55 11.30	11.35 11.35 11.25 11.45 11.45	11.35 11.20 11.30 11.25 11.20	
	Red Light + Noise					
	Periods	1	2	3	4	
Trial	S					
1 2 3 4 5		9.65 10.00 10.00 9.95 9.65	9.80 9.90 9.85 10.00 9.80	9.75 9.95 9.90 9.95 9.90	9.15 10.00 10.10 9.75 9.85	









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